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# TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE

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TRANSACTIONS

OF

# The Society of Tropical Medicine and Hygiene.

## A CASE OF ELEPHANTIASIS, TREATED BY LYMPHANGIOPLASTY.

SHOWN BY W. SAMPSON HANDLEY, M.S.LOND., F.R.C.S.

*Friday, November* 20, 1908.

J. B., a clerk, aged 46, was sent to me in April, 1908, by Dr. J. J. Udale, and I cannot do better than quote in abstract his account of the case :—

"Your communication to the *Lancet re* the restoration of lymphatic circulation in an œdematous limb has induced me to write to you respecting a patient who is suffering from sporadic elephantiasis of the left leg. He is an Englishman and has never been abroad. I have seen the case almost from the first onset in 1895. He first complained of swelling of the testicle and of pain in the leg. The limb was at that time only moderately swollen, and presented patches of erythema. The left testicle was much enlarged, hard and nodular, and the case wore the aspect of a sarcomatous growth of the testicle, with secondary glands causing the œdema. Since that time he has been to various hospitals, St. Bartholomew's, West London, Hampstead and Poplar."

In 1905 he was under the care of Sir Patrick Manson at the Albert Dock Hospital. The leg was incised, and portions of tissue removed, with some benefit. At the same time the swollen testicle was also apparently

incised. Subsequent removal of the testicle was necessary, owing to an attack of acute sepsis following the incisions. His subsequent history confirms the view that the sepsis was due, not to the introduction of bacteria, but to their presence in his tissues at the time of operation.

He still has recurrent pyrexial attacks, ushered in by a rigor and accompanied by headache. The leg during the attacks shows patchy erythema, and is the seat of pain, increasing in severity at night. While in bed the leg becomes comparatively flabby, being less tense than when he is getting about.

The patient states that the swelling began in his foot, and that he had to cut his boot. For three years after 1895 the swelling of the leg remained comparatively slight. He can walk about with difficulty.

*Condition on Admission.*—The left testicle is absent. The left lower limb is enormously enlarged, especially below the knee. On the posterior aspect of the calf are two huge masses separated by a deep cleft or crease. Another large mass occupies the dorsum of the foot. The patella cannot be felt. The thigh is much enlarged, but presents no pendulous folds. The skin is much thickened, coarse and rugose. Mr. R. Paget made nocturnal examinations of the blood for filaria without result.

On April 18, 1908, I performed lymphangioplasty,[1] the first occasion, so far as I know, that my operation has been applied to elephantiasis. Eight stout silk threads were led upwards from the foot to a point in the iliac

[1] "Lymphangioplasty: A New Method for the Relief of the Brawny Arm of Breast Cancer, and for Similar Conditions of Lymphatic Œdema," by W. Sampson Handley. The *Lancet*, March 14, 1908. See also *Archives of the Middlesex Hospital*, 1908, vol. xii., p. 28.

fossa just above Poupart's ligament. These were spaced out round the limb at intervals. Above they united to form a leash. The iliacus muscle was exposed by a short incision like that for ligature of the external iliac artery. Half the threads were passed through a portion of the iliacus muscle, and were knotted in pairs to the other four threads, thus fixing them securely to the tissues of the iliac fossa. The multiple small incisions through which the threads were introduced were closed by horse-hair sutures, leaving the silk completely buried in the subcutaneous tissues. The leg was raised on pillows.

TABLE OF MEASUREMENTS.

| Date | CIRCUMFERENCE IN INCHES | | | | | | |
|---|---|---|---|---|---|---|---|
| | Foot | 2 in. above ankle | 6 in. above ankle | Calf (maximum diameter) | Knee | 11 in. above knee | Groin |
| April 17 (before operation) | 15 | 21 | 21 | 24½ | 19 | 23 | 23 |
| April 23 (five days after operation) | 12 | 14½ | 17½ | 20 | 18 | 20¼ | 23 |
| April 27 .. .. | 11½ | 14 | 15 | 17 | 17¼ | 19¾ | 21 |
| May 2 .. .. | 12 | 15 (silk removed on May 2) | 16 | 17 | 17 | 21 | 21 |
| June 14 .. .. | 13½ | 17½ | 17¾ | 18½ | 16¾ | 19¾ | 20 |
| Normal right limb.. | 9 | 7½ | 9 | 10 | 12½ | 14 | 15½ |

At first all went well, and the success of the operation seemed almost assured. The drainage of lymph from the limb was so free that a large cushion of œdema appeared in the loin on the day following operation. This swelling extended upwards nearly to the axilla, and pitted freely on pressure. Concurrently the swelling of the leg subsided rapidly, the bandages became loose, and the pendulous folds of subcutaneous tissue shrank and took on the consistence of a sucked orange. The preceding table of measurements gives an idea of the effect of

the operation. Within five days, it will be noted, the ankle diminished in circumference from 21 inches to 14½ inches, and in nine days the calf shrank from 24½ inches to 17 inches. By April 24 the lumbar swelling had commenced to subside and was less tense.

About April 27 trouble began. The temperature, which even before the operation had not been quite steady, reaching over 100°F. on one occasion, had not subsequently exceeded 99·4° until the stitches were removed on the ninth day. All the wounds had healed except one on the outer side of the calf, from which pus could be expressed, and the one above the groin, from which clear serum exuded. On the 28th another wound on the inner side reopened and discharged pus; on the 29th another on the outer side. On the 30th there was redness along the track of the silk on the outer side of the leg. The constitutional disturbance was extraordinarily slight, and the patient only felt slight malaise, but the temperature gradually rose to 101·8° on May 2, and other wounds reopened. Retreat, if possible, was clearly imperative, and I removed all the silk, under an anæsthetic, on May 2 without much trouble, drawing it out from above after the strands had been cut across subcutaneously in the region of the foot. The tracks were syringed through with zinc chloride, 20 grains to the ounce.

At this time the case caused me not only keen disappointment but great anxiety, for pyæmia seemed a likely sequel. The suppurating tracks left by the removal of the silk were regularly syringed with antiseptics, and hot fomentations were applied. Three days after the removal of the silk, the swelling of the leg had begun to recur, although its posture remained unchanged, a fact which demonstrates that the capillary

action of the silk is the essential factor in lymphangioplasty. On May 3 the temperature had fallen to 99·2°. Subsequently the discharge of pus rapidly diminished and many of the silk-tracks appeared to close at once. On May 21 a large abscess which had formed in the thigh was evacuated. It was reported to contain *Staphylococcus aureus* in pure culture. On May 26 secondary hæmorrhage took place into the abscess cavity. The house surgeon, Mr. P. J. Chissell, very skilfully tied the external circumflex artery and stopped the bleeding. To his care and skill at this trying period the patient and I owe much. The incisions ceased to suppurate soon afterwards, and on June 11 only one incision in the leg continued to discharge. On June 19 the patient was sent to a convalescent home with the wounds entirely healed.

Up to this point the case had been a disastrous failure, but it seemed to me that hope need not be abandoned, a view which the patient fortunately shared. It was remarkable that suppuration had not set in until the ninth day after operation. If it had been due to infection of the silk from the skin during the operation, it appeared to me that it would have begun earlier. I felt very strongly that the suppuration was of endogenous origin, that it indicated a prior chronic infection of the tissues of the leg, or of the blood, by some bacterium. On June 12 I accordingly asked Dr. A. G. R. Foulerton to cultivate the blood. The result on this occasion was negative.

On August 1 the patient returned from Clacton in good health, and was re-admitted for further bacterial investigation and treatment. On August 4 a specimen of blood was drawn from the left arm, and streptococci were reported to be present in it, a startling finding,

since the pulse and temperature were normal and the patient was apparently in excellent health. Three days later further specimens of blood were taken from the left arm and from the left leg.

On August 12, with a Southey's tube, a specimen of lymph was withdrawn from the left leg and submitted to Dr. Foulerton. His report was as follows :—

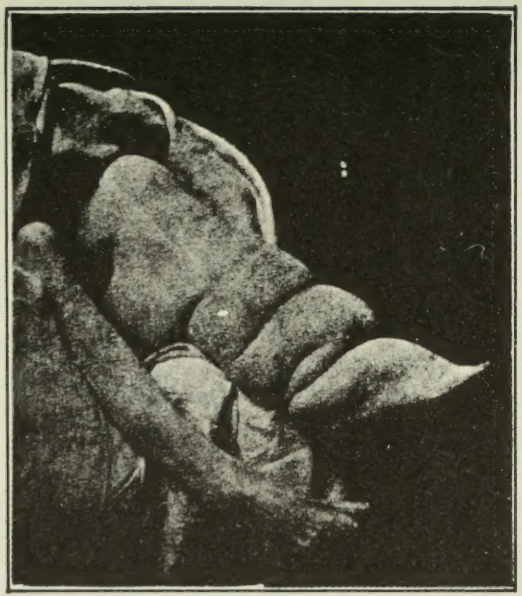
"A number of diplococci were found in films made from the lymph withdrawn from the leg, but the cultures obtained were heavily contaminated, and it was not possible to isolate the organism."

A similar diplococcus was seen in lymph drawn on August 18, and was obtained in pure culture.

The organism thus detected by Dr. Foulerton presented a remarkable similarity to that recently described by Dufougère[1] under the name of the lymphococcus, as one of the factors in the causation of elephantiasis, but I shall not enter into the bacterial side of the case, which will be described in a separate paper by Dr. Foulerton and his assistants, Mr. W. P. Hillier and Miss H. K. Whittingham.

Obviously the first thing to be done was to exterminate the infective cocci as a preliminary to further operative treatment. Dr. Foulerton prepared for me a vaccine, of which the first dose was given on August 26 (400 million cocci). Subsequent doses were given on August 31, September 8, 15, and 23, October 1 and 12. On September 11 lymph from the leg was examined, and cocci were still found in it, though they were absent from the systemic blood. On September 5 both the blood and the lymph were found sterile. The vaccine treatment had not affected the swelling, which continued slowly to increase. On September 12 a further negative examination of the lymph confirmed the result of the

[1] *L'Éléphantiasis*, Paris, 1907.



Before operation, but after vaccine treatment and rest in bed.

Eighteen days after lymphangioplasty, before treatment by massage and bandage-pressure.

previous examination. There was no diminution in the swelling of the leg.

The sterility of the tissues being thus secured, lymph-angioplasty was again performed on October 17 after most careful and thorough preparation of the skin. On this occasion the silk threads, so as to avoid the scars of the previous operation, were carried up to the gluteal region, where they terminated in the subcutaneous fat. All again went well until October 26, when the temperature rose to 99·4°. It was on this very day, the ninth after operation, that trouble had begun on the first occasion, so that the situation was alarming, although no local irritation could be seen about any of the incisions. A dose of vaccine (200 million cocci) was immediately injected, and the supports, which had raised the foot of the bed 18 inches to promote drainage, were transferred to the head of the bed. The effect of this was of course to produce a passive œdema of the left leg, the tissues of which rapidly began to refill. The limb was thus practically treated by Bier's method. Following the application of these measures, although the temperature rose to 100° on the day following, it then rapidly subsided, without the development of any local trouble. The stitches were removed on October 27 and all the incisions were found to be healed except one on the foot, where the skin was extremely thick and indolent. Any possible risk from this was guarded against by the use for a few days of weak carbolic baths for the foot. On November 1 the dressings were left off and the limb re-elevated. The remarkable effect of the operation in reducing the swelling of the limb is shown by the measurements on the next page.

Unfortunately some of these measurements are not taken at the same precise levels as those of the previous

table. It will be seen (*a*) that the limb had slowly filled again after the removal of the silk, until on October 14 its diameters were greater than before the first operation; (*b*) that within thirteen days the second operation had reduced the diameters of the leg below the minimum after the unsuccessful first operation; (*c*) on the other hand; the second operation was not so effective as the first in reducing the diameter of the thigh.

| | October 14 | October 27 | November 29 |
|---|---|---|---|
| Circumference at mid-tarsal joint .. | 14½ | 11½ | 10⅝ |
| ,, above ankle .. .. | 21¾ | 13½ | 11½ |
| ,, of leg below knee.. .. | 25¾ | 17 | 14¾ |
| ,, at knee-joint .. .. | 20 | 16¾ | 15⅞ |
| ,, of thigh 9 in. above knee | 24¾ | 21¼ | 19¼ |

(Second operation of lymphangioplasty on October 17, 1908.)

The last point is thus accounted for. At the first operation the silks were evenly distributed round the thigh. At the second operation attention was concentrated on the leg below the knee. Above the knee all the threads were led upwards in a single leash at the back of the thigh, leaving that part of the thigh to be dealt with subsequently if necessary. The argument, that the results are due to posture, rather than to the capillary action of the silk, is thus effectively met.

Sir HAVELOCK CHARLES thought the case exhibited was an exceedingly interesting one, but he did not look upon it as a cure. The limb had not yet returned to its ordinary size, and there was very great thickening of the tissues. The patient was much better than he was originally, but he did not consider that in India it would be looked upon as a cure. The method Mr. Handley had described was an excellent one, which was particularly

applicable in cases of mammary cancer and thickening of the arm. He thought that the absence of filaria was an interesting point, because, as they all knew, in cases of elephantiasis of the scrotum it was a very rare thing indeed to get filaria, but the present case was an instance where micrococci were present in the blood. He suggested that the cause of elephantiasis of the scrotum was a peripheral lymphangitis due to certain organisms. In the particular case under discussion, there were micrococci in the blood, and so long as they were present the patient did not get better, but when the vaccine was applied and the micrococci were destroyed an improvement followed. Was it a feasible explanation to suggest that the improvement in the limb was at present due to the destruction of the micrococci? In the treatment of the limb he personally should prefer a more radical method which would give a thorough result, *i.e.*, a circular incision above the knee, above the level of the diseased tissues; a vertical incision down the median line in front to the termination of the interdigital folds; a similar one on the back of the limb right down to the heel; one passing around the foot at the upper margin of the sole, leaving absolutely intact the sole of the foot and the pulps of the toes, removing the whole of the tissues right down to the deep fascia, and grafting with large grafts. The operation was quite feasible; all that was necessary was absolute asepsis. He suggested that operation to Mr. Handley in his next case. Nevertheless he thought Mr. Handley was to be highly congratulated on the result, but what struck him most was the destruction of the micrococci.

The PRESIDENT thought that, whatever the result might be in regard to the treatment of elephantiasis, Mr. Handley was to be congratulated on introducing a

new idea into surgery, and he hoped, whether it succeeded in elephantiasis or not, the mechanical idea of drainage between two different areas of connective tissue might have its application in other departments of surgery. Tropical medicine had supplied general surgery with many new ideas; it was refreshing to find that general surgery was now repaying tropical medicine.

Mr. HANDLEY, in reply, after thanking the President and Sir Havelock Charles for their kind remarks, desired to point out that it was only a month since the case was operated upon, and doubtless there was much thickening of the tissues still left. He therefore thought it was not fair to say it was not a cure, because one did not know how far the process of involution in the tissues of the limb might go.

Sir HAVELOCK CHARLES said he understood the case was presented to the Society as a cure.

Mr. HANDLEY replied that the case was not presented as a cure accomplished, but as a cure in process. That was his view of the case; and he hoped he was not wrong.

Sir HAVELOCK CHARLES said he desired to withdraw any remarks he had made with reference to that particular point, but when the patient began to walk there would perhaps be an increase of the present swelling.

### DESCRIPTION OF BILHARZIASIS SPECIMENS.

Dr. F. M. SANDWITH described a number of specimens which had been sent to him from Egypt. The first was a very good sample of kidneys, ureters and bladder, presented to the Museum of the London School of Tropical Medicine by Dr. Kartulis, of Alexandria, a Fellow of the

Society. The Fellows knew perfectly well that the kidney was not primarily affected by the eggs of the bilharzia worm. The ureters and the bladder, on the contrary, showed marked lesions. The kidney was often affected in a secondary way, because when there was any obstruction in the bladder or in the ureters, what used to be called surgical nephritis followed, hydronephrosis, pyonephrosis, and sometimes at a *post-mortem* examination the whole kidney was found converted into a thin sac of pus. He thought the Fellows would agree that these ureters were as perfect specimens as could be hoped for. The reason they were so good was because the whole of the ureter was affected. There was hardly a part of the ureter, except right up in the kidney, which was not full of little new growths of bilharzial disease, every one of which was full of eggs. The bladder itself was also a good specimen because of its thickness and the very large number of papillomata that were to be seen. It would be remembered that disease of the bladder usually began in the trigone. The kidney itself was not so interesting, although there was some dilatation of the pelvis.

The second bladder was exhibited at the Exeter meeting of the British Medical Association by Dr. A. R. Ferguson, of Cairo. He (Dr. Sandwith) brought it on purpose to contrast it with the other bladder, because it presented the most advanced overgrowth of the bilharzial disease in that organ that it was possible to have. It would be observed that there was hardly any place left for the urine to collect in. The whole viscus was distorted and put out of shape. There were some small papillomata such as were to be seen in the ureter, and some huge lumps. Not only was the bladder extremely thickened, but there was an enormous overgrowth, which, both clinically and histologically, was locally malignant.

The exact relationship between it and cancer had not been worked out, but he hoped Dr. Ferguson would do it some day.

He next exhibited a liver showing well-marked bilharzial cirrhosis, a perfectly distinct disease. He had seen such a thing half a dozen times, and at the Tropical School there were specimens of two different livers, both presented by the Egyptian Government School of Medicine. It was a real cirrhosis, though there was no prominence of the liver lobules nor retraction of the capsule as seen in other forms of liver cirrhosis. The section of liver presented an increase of new white fibrous tissue, so that its cut surface looked as if some white clay-pipe stems had been thrust into it, and it was full of eggs. The newly-formed connective tissue seemed to be confined to the neighbourhood of the portal veins, and a large branch of the hepatic vein was unaffected.

Dr. Kartulis had also sent a good specimen of the rectum showing large papillomata. It was easy to understand from its appearance how it was quite possible for the uninitiated to confuse the condition with hæmorrhoids and even with dysentery, because it produced much the same symptoms, *i.e.*, blood, mucus, tenesmus and many motions. The specimen was a typical case, although not nearly so advanced as the cases shown in the pictures exhibited, where there was a good instance of papillomata of the descending colon.

Under one microscope there was a slide showing a mass of eggs in the bladder. Those who had not worked at the subject would be interested in seeing how they were grouped together; there were literally hundreds in the field

Under the next microscope, with a higher power, there were some similar eggs from a ureter showing the miracidium within the shell.

Under the third microscope there were some bilharzia eggs from the lung. There were very few of them, there being only one or two in a field, and perhaps only twelve in the whole slide. The only interest in showing the slide was because of the rarity of bilharzial disease in the lung. Evidently the female that laid the eggs had lost her way and had not gone to the bladder and the rectum as most of the females did. Dr. T. S. Kerr had also very kindly sent a specimen of lateral-spined ovum from Porto Rico, which was to be seen under the fourth microscope, while under the fifth were eggs on a warm stage, from which living miracidia could be seen to emerge. These eggs were obtained from urine from Haslar, kindly supplied by Fleet-Surgeon Bassett-Smith.

There was also on exhibition a series of photographs of bilharzial disease, including some illustrations of how the disease caused prolapse of the rectum, and there were also some instances of huge cauliflower excrescences of the vulva in cases where the urethra and the bladder were apparently normal in the female. Then, lastly, there was shown a model of a vesical calculus from a case described by Mr. Owen Richards in the *Lancet* of July 25, 1908, now on its way to the Museum of the London School of Tropical Medicine. By its side was a model of the largest stone ever successfully removed, a feat accomplished by Mr. Milton in 1893, the stone weighing 34½ oz. Mr. Richards's case was the second largest, weighing 31·4 oz. Mr. Milton's stone was removed by abdominal section, because it was impossible to remove it by lithotrity or by suprapubic operation; while Mr. Richards's stone was extracted piece-meal by osteotome and mallet above the pubes, aided by perineal section. He tried to perform lithotrity, but failed to do so because Milton's 5-inch lithotrite was not large enough

for it, and the stone was therefore broken up and extracted in pieces. Subsequently the broken pieces were put together in order that a cast might be made. He thought the Fellows would all agree that the originals of the two stones were in their proper place, namely, in the best pathological museum in the world, in Lincoln's Inn Fields.

Discussion.

Dr. Harford enquired whether Dr. Sandwith could state in what proportion of cases evidence of bilharziasis disappeared. He was very much interested in the subject, because he had seen that day the wife of one of the missionaries of the Church Missionary Society who came home from East Africa with the repute of having had bilharzia in the bladder. She had passed urine and also a calculus which contained bilharzial eggs, and afterwards she had had mucous colitis. When she came home no ova could be discovered at all, so that it could not be said that the mucous colitis was associated with the bilharzial disease. Since she came home, about eighteen months ago, she had not had any sign of diarrhœa or any other bad symptom; and he therefore would be very much obliged if Dr. Sandwith would state his general view of the case, and whether the trouble was likely to appear again.

The President asked Dr. Sandwith whether so-called bilharzial cancer of the bladder was ever associated with secondary growths; and also whether the ova in the liver were terminal-spined or lateral-spined. The Fellows might remember that some time ago a discussion occurred at the Society on the subject of the specific differences between the lateral-spined ovum and the terminal-spined ovum, and some of the Fellows then maintained that the two were distinct species. Quite recently a paper had

been received at the School of Tropical Medicine from a Brazilian observer, who described in detail some cases of rectal bilharzia, in all of which the ova were lateral-spined; and although he had investigated the urine of hundreds, if not thousands of Brazilians, including the cases of rectal bilharzia, in no instance did he find bilharzia in the urine, and in no instance had he ever found a terminal-spined ovum, bearing out the contention that the parent of the lateral-spined ovum and the parent of the terminal-spined ovum probably belonged to different species. He would be glad to know if Dr. Sandwith had any recent information bearing on that subject.

Dr. SANDWITH, in reply, said he was very much obliged to Dr. Harford for putting his question in the way he did. The general way of asking such a question was "How soon will the patient recover?" But Dr. Harford had been bold enough to say that the patient had recovered, and asked what guarantee was there that the patient would not have a relapse? He imagined from what Dr. Harford said to him before the meeting that he referred to the case of people who had lived in a country where bilharzial disease was endemic, and came home to a country like England, where there was no bilharzial disease, and therefore no fear of fresh reinfection. In those cases he maintained that they all got well if they lived a sufficiently long time in a country where there was no bilharzial disease. That meant in point of fact that the parent worm was not eternal, that it would not live for ever, although apparently it could live for a great number of years. He began by thinking, judging from the cases that he had seen, that the patients lost the eggs of the bilharzial disease within two years. He confessed, however, that he had now had to extend the time to four

years, and cases had been published where people, even after ten or fourteen years, had been known to pass bilharzial eggs, especially after exertion such as riding. He advised Dr. Harford's patient not to have her urine examined under the microscope immediately after some prolonged exertion, such as bicycle riding, horse riding, a long walk, or something of that kind. He knew from experience that several young Englishmen contracted the disease in Egypt and lost it within a couple of years. He hoped Dr. Harford's patient had lost the disease, and it would remain absent unless she subjected herself to some great fatigue. The President had enquired whether the condition which was seen in the bladder given by Dr. Ferguson was cancer followed by secondary manifestations. Speaking generally, that was not so, the case had no secondary manifestations. That was one of the interesting points, because one would expect so much irritation in the bladder to give rise to the occurrence of cancer. When cancer did exist in a bilharzial patient (and one native out of three in Lower Egypt had bilharzia), there was no very great probability that the cancer had been produced by the bilharzial irritation. The President asked a question about the liver; and he (Dr. Sandwith) asked to be allowed to read a note by Professor Symmers of the microscopical examination of such a liver: "There is a great increase of the periportal connective tissue of the sublobular and larger portal canals, the fibres of which are mostly wavy and parallel, but here and there arranged concentrically, surrounding ova. Eggs found in the liver consist usually only of the shell with lateral spine." That was the answer to the President's question, that they had a lateral spine, *i.e.*, that they belonged to the rectal form rather than to the bladder form. They were generally shells; he could not

show a perfect specimen under the microscope because the shells were destroyed in cutting sections. No eggs are seen lying free among the hepatic cells, nor usually in the larger blood-vessels which permeate the new cirrhotic tissue. The whitish nodules on the surface of the liver consist of fibrous tissue containing eggs. The President had also told them of the Brazilian cases, which confirmed previous knowledge, that in the rectum the lateral-spined egg was obtained and not the terminal-spined egg. Many hundreds of cases had been examined by Bilharz and Griesinger and ever since their day and that seemed to be an invariably true rule. Wishing to avoid a debatable point, he had desired to confine his remarks that evening to the terminal-spined eggs until Dr. Kerr offered to bring forward a specimen of lateral-spined ovum. The whole question had been extremely well summed up by Professor Looss in the July number of the *Annals of the Liverpool School of Tropical Medicine*, to which he (Dr. Sandwith) was unable at present to add anything.

# THE SIGMOID FLEXURE AND RECTUM IN POST-DYSENTERIC DIARRHŒA, SPRUE, AND MUCOUS COLITIS.

By JAMES CANTLIE, M.A., M.B., C.M., F.R.C.S.

Before dealing with the pathological changes met with in the mucosa of the rectum and sigmoid flexure, I would first draw attention to several important points in the anatomy of these portions of the alimentary canal.

Examinations of the rectum by the sigmoidoscope presented to me so many seemingly irrelevant anatomical and physiological aspects that some years ago I proceeded to examine the rectum and sigmoid flexure more closely anatomically, and to review the functional activity of these parts.

My attention was first directed to the junction of the rectum and sigmoid flexure. I am well aware that the name "sigmoid flexure" has been well-nigh eliminated from modern anatomy, but for convenience of description I adopt the term in its time-worn significance.

On passing the tube of the sigmoidoscope up to and through the junction of the rectum and sigmoid one is struck by two points: When the sigmoidoscope tube approaches the junction of the rectum and sigmoid the bowel is found (*a*) to narrow in calibre, and the walls to lie in apposition, quite different from the wide and easily distended channel of the rectum below; (*b*) the calibre narrows to an almost rounded aperture resembling in appearance the os uteri, and still further is the resemblance confirmed by a normal protrusion of this portion of the bowel into the wider channel of the rectum immediately below, just as the uterus projects into the wide

vagina. There is here a normal prolapse, in fact, of the sigmoid into the rectum, to which, as we shall see, a functional activity belongs. As the sigmoidoscope approaches and is passed through the junction of the rectum and sigmoid the patient complains, even when the bowel is healthy, of an untoward sensation, which is always attended by some remonstrance, and frequently causes the patient to exclaim, "I am afraid I am going to have a motion." In all departures of the bowel from the normal state the patient experiences pain at this junction so acute at times that the attempt to traverse the gateway from the rectum to the sigmoid has to be given up.

These two points so interested me that I set to work to examine the bowel at this point, and for the purpose I examined 200 *post-mortem* specimens taken at random as to age and sex from the *post-mortem* room.

## Anatomical Study of the Sigmoid Flexure and Rectum.

On slitting open the lower 2½ ft. of the large bowel, the following points are to be noted: The sigmoid flexure is, except in cases of abnormal distension due to chronic constipation, of markedly narrow calibre, terminating abruptly above at its junction with the wide descending colon, and still more abruptly below at its junction with the rectum.

At these two points the mucous membrane loses its folds, the submucous tissue is thinned, the circular muscular fibres are thickened, and the longitudinal fibres of the gut approach and actually touch each other. These features are more pronounced at the lower end of the sigmoid than at the upper. For these

two apertures, from their resemblance (especially the lower) to the pylorus between the stomach and duodenum, I have proposed the names *Colo-sigmoid pylorus* and *Sigmo-rectal pylorus*, or the upper and the lower pylorus of the sigmoid flexure.

There is no prolapse of the colo-sigmoid pylorus into the lumen of the sigmoid, but there is in all healthy intestines a distinct prolapse of the sigmoid into the rectum below. The anatomical significance of these two points—the entrance and exit—of the sigmoid flexure, and the physiological purpose of this part of the intestine, become manifest, but hitherto little or no attention has been paid to them.

It is evident that the fæces are prepared for excretion in the sigmoid flexure, losing their fluid character and gaining shape before reaching the upper part of the rectum. The desire to go to stool is declared when the fæces pass the sigmo-rectal pylorus, and the necessity to go to stool is present from that moment. The fæces are retained in the rectum by will power directed to the anal sphincter until such time as it is convenient to empty the rectum, or until it is rendered necessary, owing to rectal spasm. The confirmation of this function of the sigmo-rectal pylorus is evident when the sigmoidoscope is being introduced, as already indicated, the patient declaring that a stool is passing when the tube of the instrument passes through the pylorus.

The fact that the sigmoid protrudes or prolapses into the upper end of the rectum no doubt adds to the functional power of the pylorus, for a larger mass of circular muscular fibres is called into play than would be the case were the tube straight, with no involution.

The sigmoid flexure is no mere tube for transmission of the contents of the bowel; it is endowed with functions

dependent in part on anatomical construction and in part on physiological processes provided for by the nature of its anatomy. Until fæces reach the sigmoid they are normally of a more or less pultaceous consistence, but when they emerge from the sigmoid into the rectum they have attained greater consistence and solidity.

## Specific Structural Anatomy of Rectum and Sigmoid.

(*A*) *The muscular fibres.* (1) *The longitudinal fibres.* When the lower 2 ft. of the intestine, from the descending colon to the anus, are examined closely, it will be found that the widely separated longitudinal muscular fibres of the colon approach one another at the upper end of the sigmoid flexure (colo-sigmoid pylorus) until two at least of the bands touch each other laterally. On the sigmoid flexure itself the bands again separate, until the junction of the sigmoid flexure with the rectum is reached (sigmo-rectal pylorus), when the bands of longitudinal fibres approach one another closely and, in many (if not in all) instances, muscular fibres interchange and pass from one band to another. In the rectum at the upper part the longitudinal muscular fibres again separate, to meet towards the lower end of the rectum.

(2) *The circular fibres* of the descending colon at the lower part gradually become more closely packed, and at the upper end of the sigmoid (colo-sigmoid pylorus) assume the character of a thickened ring. In the sigmoid flexure itself the circular muscular fibres are apparently more highly developed than in any other part of the large intestine, with the exception of the narrow ring-like structures at the commencement and end of the sigmoid. At the junction of the sigmoid flexure with

the rectum the circular muscular fibres are closely applied and hypertrophied, forming a ring narrower than at the upper end and possessing more exactly the appearance of the gastro-duodenal pylorus. This is markedly *the* pylorus of the sigmoid flexure (the sigmo-rectal), and is endowed with physiological functions of great importance. In the rectum the circular fibres are scattered in the upper part of the rectum, allowing of some sacculation, but in the lower part they become closely applied, more especially at the internal sphincter.

(*B*) *The Mucous and Submucous Membranes.*—On laying open the lowest 2 ft. of the intestine from anus to colon the appearance of the bowel is found to be (*a*) a narrow tube-like passage at the lower 4 in. of the rectum in which the mucous membrane presents a transversely corrugated appearance. (*b*) In the upper 4 in. of the rectum the gut assumes a sacculated appearance and is capable of wide dilatation. The arrangement of the folds of this portion of the tube, and of the mucous membrane in particular, shows that it is subject to puckering of its calibre, as no doubt it is, owing to the contraction of the levator ani during the act of expulsion of the fæces, when the pressure from above is aided by the levator ani pulling the rectum on to the mass of the fæces as a glove is pulled on to a finger.

(*C*) At the junction of the sigmoid flexure and rectum the bowel is narrowed, the mucous membrane is smoother, the submucous membrane is more dense than elsewhere, and, os uteri-like, it projects into the lumen of the tube. Here is a normal narrowing and a normal prolapse of the intestine; a spot which physiologically is endowed with the function of the natural retention of the fæces, of preparing the contents for expulsion, and as the dial of indication of the necessity for defæcation. Pathologically

it is the most important part of the lower bowel and is worthy of a distinctive designation, and I have therefore bestowed upon it the name of pylorus of the rectum, to distinguish it from the less important gate-way at the upper end of the sigmoid flexure—the sigmo-rectal pylorus.

(*D*) Above the sigmo-rectal pylorus is a decided pouching of the sigmoid flexure for an inch or less; and from hence upwards the mucous membrane is here and there corrugated transversely, with smooth areas between.

(*E*) At the upper end of the sigmoid flexure the bowel is narrowed, presenting the upper or colo-sigmoid pylorus, which is a repetition of the lower or sigmo-rectal pylorus in structure and appearance, although the narrowing is less, the prolapse or involution not marked, and the submucous coat less dense.

(*F*) Immediately above the sigmoid flexure the descending colon is sacculated, and the mucous membrane frequently pigmented.

## The Introduction of the Sigmoidoscope, or Proctoscope into the Bowel.

*Position.*—When possible the patient should be placed on knees and elbows, but the examination can be conducted while the patient lies on the left side with the knees well drawn up and the buttocks projecting over the edge of the bed or table.

It is inadvisable to use an anæsthetic, as the patient's sensations are a useful aid in preventing dangerous force in the introduction of the metal tube. Having warmed and vaselined both tube and anus, pass the sigmoidoscope tube through the anus and push it onwards for 1½ in. in a direction rather forwards, then backwards for next 4 or 5 in. along the hollow of sacrum, when it is necessary

to direct the point forwards again and push onwards in a line from the anus to the umbilicus. The introduction has to be done gently, and the point is made to trend towards the point of least resistance with frequent cessations, alterations of position of point, and waiting until any painful or uncomfortable sensations subside. As the sigmo-rectal pylorus is touched there is obstruction to the passage of the instrument, and the patient calls out owing to discomfort or pain. It may be merely the feeling that a stool is coming, or it may be that distinct and even acute pain is experienced. The blunt cone of the instrument feels as if it had come against a wall of dense obstruction, so that it is hopeless to pass the instrument further. This may prove, however, not to be the case, for by a little patience and coaxing the instrument may be made to enter the sigmoid flexure, and, as it does so, the feeling that it has passed over a roughened or tightened spot will always be felt. The blunt-ended stylet may now be withdrawn and the electric light introduced.

### Situation of Lesions in the Rectum and Sigmoid Flexure.

The lesions in the bowel may be extra-intestinal, endo-intestinal, or intra-intestinal; that is, without, in the substance of, or within the bowel.

(1) *Extra-intestinal.*—Pressure upon the bowel from without, from a pregnant uterus, uterine fibroid, retroverted or retro-flexed uterus, ovarian cysts or other ovarian ailments, tumours originating in or finding their way into the pelvis, aneurism of the left iliac arteries, tumours of the left kidney, and any form of inflammatory matting of peri-rectal tissues, may cause intestinal casts to be shed and passed at stool, or mucous catarrh of the

intestine above the seat of the pressure and passing of gelatinous-looking material continuously or intermittently.

(2) *Endo-intestinal.*—Stricture of the bowel, malignant or benign, occurs most frequently at the junction of the sigmoid flexure and rectum. Stricture at this situation follows the rule of tendency to stricture in the alimentary canal generally from mouth to anus, for wherever a wide portion of the canal joins a narrow, or a narrow expands into a wide portion, there stricture is liable to occur.

(3) *Intra-intestinal Lesions.*—Abrasions, excoriations, granular surfaces, ulcers, œdema, cicatrices, and acutely tender spots.

### Situation of Lesions in the Lower Two Feet of the Lumen of the Bowel.

Neglecting actual anal lesions and piles, the following may be seen in cases of colitis and post-dysenteric conditions :—

(1) Tropical ulcer of the rectum. On the posterior wall of the bowel, over the tip of the coccyx, as a rule, a common form of ulcer is met with in tropical patients who have suffered from intestinal trouble. The ulcer is small, single, exquisitely painful when touched, and causes great pain after defæcation, which lasts from half an hour to one and a half hours, or more. It causes the neuralgia of the rectum not infrequently met with in the last stages of sprue. The cure is simple. A scalpel drawn across the ulcer longitudinally and to the depth of ¼ in. will cure the condition.

(2) About 2 to 3 in. up when multiple ulcers (or cicatrices) are seen with irregular outline of varying size, with the long axis as a rule transversely to the lumen of the bowel, with dull red margins and surfaces covered with greyish or brownish coloured material, and not

markedly painful, tertiary syphilis will be found to be the cause. These will disappear with appropriate anti-syphilitic treatment.

(3) When the sigmo-rectal pylorus is approached the lesions associated with colitis, post-dysenteric states, and chronic diarrhœa are met with. These may be: (*a*) Œdema of the part. (*b*) Congestion and narrowing of the lumen of the tube. (*c*) A velvety or granular appearance of the mucous membrane, with a narrowing of the bowel. (*d*) Multiple points of small vesicles filled with purulent matter in groups on the bowel wall. (*e*) Ulcers, multiple as a rule and small in area, each almost the size of a split pea, with pink margins, raised edges, purulent matter on the surface, and usually very painful when touched. (*f*) Cicatrices of old ulcerations with puckering of the bowel wall, the edges of which bleed easily when the sigmoidoscope is passed. (*g*) When there is a recurrence of intestinal flux in old-standing cases of colitis or dysentery, several of the above conditions may concur. There may be œdema with granular mucous surfaces, old cicatrices with hæmorrhagic base, and several ulcers or abrasions. (*h*) Stricture of the bowel, in consequence of the irritation due to cicatrices, ulcers and chronic inflammation, is apt to ensue.

### Congestive or Fibrous Thickening of Bowel the Cause of Recurrence of Symptoms of Colitis, Dysentery, or Diarrhœa with Mucus.

The condition of bowel noted above, which occurs at the sigmo-rectal pylorus, results in thickening of the bowel wall. The thickening may be more or less of a permanent nature, and becomes aggravated occasionally by local congestion. Whilst the congestion exists the

fæces are at first retained and then an intestinal flux develops, consisting at first of fluid fæces, accompanied after a few days by the presence of mucus in quantity in the stools. The congestion and œdema of the sigmo-rectal pylorus may subside, the diarrhœa and mucus lessen, and fairly solid stools, accompanied by gelatinous material in greater or lesser quantity, go on for some time, in fact until the next recurrence or until the condition is cured.

The narrowed strictured condition of the sigmo-rectal pylorus, however, remains, and each recurrence of intestinal flux marks a further development in the thickening of the bowel. The explanation of mucous casts, of mucus in the stools, of gelatinous exudation preceding, with, or after a motion more or less solid, is readily understood, and the recurrence of intestinal flux at intervals of a week or a month is explained away when the local condition of the gut at the sigmo-rectal pylorus is recognized.

### Treatment.

With the recognition of the cause the mode of treatment is apparent: cure the strictured condition and the ailment disappears. In the bowel we have a condition exactly parallel to a stricture of the urethra as regards late symptoms. In the urethra a stricture is attended by a narrow point in the canal behind which a drop of urine lies, it sets up a catarrh of the mucous membrane, which in time occasions a discharge which flows backwards to the bladder and appears in the urine or overflows the strictured wall and appears at the urethral orifice; occasional attacks of œdema and congestion aggravate the stricture, causing obstruction to micturition or temporary retention. In the rectum similar conditions obtain.

Given a congestion and narrowing of the sigmo-rectal or colo-sigmoid pylorus, there is a pocket behind (above) the stricture which becomes catarrhal from some fæcal retention; the catarrh occasions abraded surfaces, the discharge, at first watery, becomes later muco-purulent, then mucous, and finally gelatinous, in which state it is maintained until the condition is cured.

(*A*) When the cause of the intestinal shedding of mucus from the lower part of the bowel is due to extra-intestinal causes, these must be removed if possible. Tumours must be dealt with, aneurism treated, and such conditions as are irremovable must be ameliorated.

(*B*) When the bowel is strictured by endo-intestinal strictures, these must be treated by one or other of the well-established plans.

(*C*) When the lesion giving rise to the narrowing of the bowel is from intra-intestinal causes, the rational treatment of the condition, if the above facts have been studied and grasped, is apparent. Cure the strictured condition of the bowel, for by this means only will success result.

(1) If the narrowing of the bowel is merely congestive with some œdema of the mucous membrane and occasional ulcers, drugs and enemata may in time effect a cure. By keeping the motions soft with drugs, the irritation caused by the passage of fæces over the tender spot in the bowel is lessened. Ipecacuanha (with or without emetine) in 20-grain doses given daily for a week, and then in 10-grain doses for fourteen days, will allay the catarrh above the narrowed part as no other drug will. Combined with drugs, enemata are useful. I find sea-water by far the best enema for the purpose. Two pints of warmed sea-water (not diluted), thrown slowly up the bowel and retained as long as possible, soothe the bowel

and bring away the mucus in quantity. The enemata should be given every second day for ten days, and then twice a week for ten days following. An occasional enema, say once a week or fortnight, may with advantage be continued for some months. When sea-water cannot be had, sea-salt may be utilized instead.

(2) When the surface of the bowel in the neighbourhood of the sigmo-rectal pylorus is ulcerated, it is usually sufficient to touch the surface of the ulcers with pure carbolic, introduced in a stylet through the tube of the sigmoidoscope on a small piece of cotton-wool. One or two applications, combined with the treatment recommended above, will speedily diminish the pain attaching to the ulcerated surfaces and heal the ulcerations.

(3) When the chronic diarrhœa is of long standing, accompanied by the recurrence of intestinal flux, with mucous and gelatinous discharges from the bowel, and all the other symptoms of a narrowed gut, the only treatment is to cure the strictured part on the same principles and by similar methods as in the case of the urethra. To accomplish this, the tube of the sigmoidoscope must be introduced up to the strictured part and an india-rubber (œsophageal) tube passed through the stricture gradually and with gentle pressure. This is by no means an easy matter, even with the help of the sigmoidoscope and with the advantage of seeing the end of the tube whilst introducing it through the orifice.

The point of the tube is apt to become deflected and pass down between the instrument and the bowel wall. If this is the case when introduced under such favourable circumstances, what must be the case when a long tube is passed up the bowel in the ordinary way? The fact is, that a long tube passed up the intestine in non-strictured bowels reaches the sigmoid flexure, even in skilled hands,

in a very small percentage of instances; so small that when one reads that the doctor "washed out the bowel by a long tube introduced 2 or 3 feet" one takes the statement with all reserve, knowing from X-ray photographs that the tube coils up in over 90 per cent. of instances in the rectum and never reaches the sigmoid flexure at all. Unless the œsophageal tube is made to pass thoroughly through the stricture cure will not result; and the failure of this treatment is due to only one cause, namely, the failure to pass the œsophageal tube through the strictured part. The œsophageal tube should be introduced twice weekly for the first fortnight, afterwards once a week for a month, and once a fortnight for one or two months more. Combined with the passage of the tube, the treatment by ipecacuanha or cyllin, and by sea-water enemata, should be continued.

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## Discussion.

Sir Havelock Charles thought the Fellows were very much indebted to the author for the very able paper he had read. He would have liked to hear Mr. Cantlie go more definitely into the anatomy of the rectum, because he was rather hazy at present what Mr. Cantlie meant by the rectum, since he had been adopting the somewhat antiquated description of the gut now given up in modern anatomical schools. The rectum begins at the termination of the pelvic mesocolon—that is, about the level of the third sacral vertebra. The part of the colon from this point *upwards* to the brim of the true pelvis is now known as the *pelvic colon*, and corresponds to the portion of the "sigmoid flexure" which lies in the true pelvis, together with the so-called first part of the rectum. It is this portion which varies so

much in length—it may be as short as 5 in. and as long as 35 in. Here we have a very practical point as regards the use of the "long tube." or any such appliance; in the former case there is no difficulty, in the latter it is a practical impossibility. The gut extending from the brim of the true pelvis to the crest of the ilium, which lies in the iliac fossa, is the *iliac colon*. Where it joins the pelvic colon, and where this latter passes into the rectum proper, there are often narrowings of the tube, though such a thing as Mr. Cantlie's pelvic pylorus the speaker had never seen. Mr. Cantlie speaks as if Nature had made no arrangement for easing the weight of fæcal matter from off the sphincter. He has forgotten the three or more *lateral inflexions*, which are marked on the exterior by creases, and present on the interior of the rectum as crescentic shelves. These are the rectal valves of Houston, and they help to support fæcal contents, the flexions increasing the lateral capacity of the gut. Under pathological conditions their concave margins are liable to ulceration. It is possible that under certain conditions the loose mucous membrane from above might present in the manner suggested by Mr. Cantlie as resembling an os uteri, the valve itself being the suggested "pylorus." These valves are to be found in the fœtus—thus pointing to their evolution to suit our change from the quadruped to the biped, in long by-gone days, and the consequent necessity for a support for fæcal matter. In the dissecting-room students often fail to see these well, especially if the guts are not fresh. But in the frozen body and live subject they are very manifest.

The tropical ulcer does not differ from our old friend the painful ulcer of the anus, so well described by the late Mr. Hilton, whose method of treatment gives relief.

When medication to the upper part of the rectum is indicated, or when the pelvic colon is involved, the step of most importance is a thorough dilatation of the sphincter ani. It facilitates examination by direct and reflected light, proper local medication, and confers the boon of physiological rest by the abolition of reflex spasm. In itself it has a great curative influence.

These remarks are not made in a carping spirit, but only that Mr. Cantlie may shed more light on an interesting subject.

Mr. CANTLIE, in reply, said that before he read the paper he was afraid of the reception it would receive, because when one brought forward a subject that was out of the ordinary one had to be prepared for a great amount of criticism. Sir Havelock Charles, however, had spoken so kindly that he had encouraged him to go on investigating the matter still more closely. He had searched for the folds of Houston many a time. Every time he had passed a tube he had had in his mind the folds of Houston, but he could not say he had ever recognized them clinically. Anatomically he could quite understand the difficulty of finding the folds. It was not easy to find them in the dissecting-room unless they were most carefully looked for, and in fresh bodies. He believed all practitioners recognized the dilatation of the sphincter as one of the very first things to be done when any trouble occurred which could not be cleared up otherwise. Piles were often associated with chronic diarrhœa or the dysenteric state which he had tried to describe. Recently he had been insisting upon the dilatation of the rectum, and it was astonishing how the trouble then disappeared. He had referred to the application of carbolic acid to the surface of the ulcerations.

Another thing he had used, which perhaps some of the Fellows might look upon as a fad, was sea-water. In his experience there was nothing so satisfactory in bringing away the mucus as sea-water—not salt and water, nor Tidman's salt and water—which was better than salt and water. Sea-water itself seemed to be the best thing which could be used as a wash for the rectum; it was most effective in washing out mucus from the intestines. In addition it seemed to allay irritation, to heal ulcers if they were present, and to get rid of the œdematous condition better than anything else he had tried. Sea-water could be easily obtained in London from the Great Eastern Railway Company. After another two years' work on the subject he hoped to be able to bring it forward again, and to place before the Society what was right and wrong in the matter.

# THE PRESENT POSITION OF THE RAT-FLEA THEORY OF PLAGUE; RECENT OBSERVATIONS IN CALCUTTA.

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(*Friday, December* 18, 1908.)

*Introductory.*—Doubtless to many of you the rat-flea theory of plague—that is to say, the explanation of human plague as due to the bites of plague-infected rat-fleas—is one of the established facts of modern medicine. In short, it is rapidly acquiring the status of a text-book dictum, as exemplified in Bullock's article in Clifford Allbutt's "System of Medicine," Roger's "Tropical Fevers," and, with reservations, in Manson's last edition of his "Tropical Diseases." By most of the medical journals the theory is accepted without any reserve at all. In fact, there is a certainty that unless the other side of the question is promptly and vigorously put forward, a stage will be reached where the matter will be practically beyond discussion. This stage I am anxious to anticipate. My reason for doing so is that my knowledge of plague is based on ten years' experience of practical plague work—that is to say, of carrying out measures framed in accordance with accepted theories. Unfortunately, amongst my experiences is that of devoting all my energies to the carrying out of measures which I knew to be comparatively futile, the futility of which I subsequently demonstrated up to the hilt by an experiment on an enormous scale. I refer to disinfection with 1 in 500 acid perchloride of mercury, under Oriental conditions. Let it

be remembered that measures based on a wrong theory necessitate not only the waste of hundreds of thousands of pounds of public money, but also the impossibility of following out other lines of action which seem to promise definite beneficial results. As an example, demolition of infected centres was deferred in Calcutta for fully two years owing to the prevalence of erroneous ideas as to the value of disinfection.

To bring home to you how intimately the theory of plague is connected with practice, an incident in Calcutta may be given. The Chairman of the Corporation had just listened to a lecture in which the current *Mus rattus* theory of plague had been given due prominence. His first remark after the lecture was: "According to what we have just been told, our measures for rat-destruction may do harm rather than good. The brown rat kills the black, which is the one that causes plague; and if we trap more brown than black we will cause more damage than good." It was only when he realized that the current views put forward did not represent the opinion of the lecturer that he was reassured, and went on with rat-destruction.

In view of these considerations, it will be clear that if I have any doubts as to the truth of the rat-flea theory, it is my duty not only to bring forward the facts against it, but also to call your attention to what fallacies there may be in the evidence in its favour. For, as has just been indicated, the measures on which depend the lives of millions, and the expenditure of hundreds of thousands of pounds, are framed in accordance with theories; so that no personal or other considerations can interfere if the verification or otherwise of these theories is in question. This is my justification for what might otherwise be deemed unnecessary and captious criticism.

*History of the Rat-Flea Theory of Plague.*—The most desirable mode of dealing with the subject would have been the purely chronological, but a difficulty has crept in. There are first the early stages of the theory based on the work of Yersin, Tirolia, Hankin, Nuttall, Ogata, and Simond. These I shall only mention. Next comes the middle stage of the rat-flea theory—the stage inaugurated by the epoch-making experiments of Liston with guinea-pigs in Bombay. Here we have the theory in its prime, but only indicated in Liston's pamphlet, not quite definitely put forward in print. But, though not in an authoritative print, it was to be found in many of the medical papers and in the mouths of all Indian officials—medical and otherwise. The theory then was a beautiful and splendid production, "teres totus atque rotundus." It fitted in with all the known or assumed conditions of plague. Plague was most rampant where the *M. rattus* was most prevalent; its season corresponded with the season of the maximum prevalence of rats as indicated by the breeding-season; and, finally, plague was correlated with the maximum prevalence of rat-fleas, which in turn was correlated with the habits of *M. rattus* as compared with the habits of *M. decumanus*. The former was a roof-haunting rat liable, when dead, to drop his fleas on man; the latter a sewer and burrowing rat which left his fleas on his demise in his burrows where they could not attack man.

In 1906 began the issue of the reports of the Advisory Committee, or the Commission, as I shall call it throughout. Their experiments and observations all pointed in one direction—the prepotency of the rat-flea as the cause of human plague, but no definite final conclusions on the subject were published until the production of "The Etiology and Epidemiology of Plague," a summary of the

Commission's work and conclusions. Here comes the chronological hitch. I recently read a paper before the Calcutta Medical Society in which I criticized many of these conclusions under the mistaken impression that they represented the findings of the Commission. I am now informed that this pamphlet represents not the official views of the Commission, but the personal views of the member who drew it up, Major Lamb, I.M.S. I must express my regret at the error I have fallen into in attaching to the Commission misdirected criticisms. The criticisms, of course, stand; but they should have been directed not against the Commission, but against Major Lamb. The only excuse I can give for the error is that, as far as I know, it is still widely prevalent in India.

*The M. rattus Theory.*—I have already indicated what this theory was in its prime, how it was supposed to fit in with the prevalence of *M. rattus*, with the breeding season of *M. rattus*, the habits of *M. rattus*, &c. So little doubt was there about the predominant part played by the *M. rattus* that the difference in the rat population of Bombay and Calcutta was brought forward as the explanation of the difference in the severity of attack. Bombay had a rat population in which, according to Liston, *M. rattus* predominated, and accordingly it suffered severely; Calcutta had a rat population in which *M. decumanus* predominated, and hence it was comparatively mildly attacked. I shall have to return later to the mildness of the Calcutta attack and show how very comparative it was. So I started to verify the supposed fact that *M. decumanus* was the common rat of Calcutta. A year's work showed that the principal plague rat of Calcutta was one never dreamt of before—the Bengal rat, *Nesokia bengalensis;* that *M. decumanus*,

the brown rat, also played an important part, and that the *rôle* of *M. rattus* was almost negligible. Incidentally, it was also found that rats bred freely all the year round, and that the difference of habits between *M. rattus* and *decumanus* had been exaggerated. I will not go into details, as the work of Dr. Crake and myself as to prevalence and relative habits of the different rats has been fully confirmed by Captain Gourlay, I.M.S., in Dacca. In consequence of this Calcutta work there was a re-examination of rat conditions in Bombay, with the result that the presence of the Bengal rat was verified there, too; still more unexpected was it that the commonest rat and the one which suffered most from plague was the brown rat. The exact figures, as given in the Commission's second report, were that 70 per cent. of all rats examined, and 84·6 per cent. of all rats found infected, were *M. decumanus*—the brown rat.

One would have expected after this that the black rat would have retired into a decent obscurity, but the Commission in their third report brought him to the front again and produced a modified *M. rattus* theory. They hold that the epizootic appears earlier amongst *M. decumanus* and reaches its culminating point ten days sooner than in *M. rattus*. While a few cases of plague in man may be caused directly by *decumanus* as he roams about the gullies and lower stories, it is only when plague has spread from *decumanus* to the house-haunting *rattus* that man becomes seriously infected. The explanation of the priority of the epizootic in *decumanus* is that he is more predisposed to attack than *rattus* in consequence of harbouring twice the average number of fleas—eight against four. There are many common-sense objections to this theory. What about plague in Ulmarra, in which instance, though *M. rattus*

was numerous, only *M. decumanus* played any part in connection with human plague; or plague in Calcutta, where *M. rattus* is almost a negligible quantity; or plague in the Punjaub, where *M. decumanus* is non-existent, and *M. rattus* is as much a burrowing rat as is *M. decumanus* in Bombay; or the first epidemic in Cashmere in which infection was direct from man to man and rats played no part at all? The only objection I wish to deal with is a very serious one. The chart demonstrating priority is one based on the percentage variations from the corrected weekly mean of rats collected and found infected. The crude figures on which this chart was based show, however, no priority in the *decumanus* epizootic. On the contrary, they show that the highest percentage of plague-infected rats was found in the same week in both *rattus* and *decumanus*—the week ending March 17. In that week *decumanus* showed 42·3 per cent. infected and *rattus* 32·7 per cent. infected. (Table XXIV. following p. 795, *Journal of Hygiene*, Vol. VII., No. 6, December, 1907.) At the very end of the fourth and most recent report of the Commission appears another chart of *rattus* and *decumanus* plague. The apices of the two curves here coincide exactly.

After this it may seem rather needless to take up their conclusion "that the (high) co-relationship between plague in man and plague in *M. decumanus* is probably spurious, depending on the co-relationship between plague in *decumanus* and in *rattus*." My reason for mentioning it is that it is one of the main arguments brought forward by Major Lamb in an attempt to rebut the criticisms published in the Calcutta Plague Report of 1907. The common-sense objections have already been dealt with; there are also most serious technical mathematical objections. Rats were not collected uniformly through-

out the year. In the height of the epidemic season the average collected per week was nearly 5,000. In the slack season it averaged only about 1,300 per week, with a minimum of 961. Again, the total rat population of Bombay can hardly be estimated at less than 5,000,000; that is to say, the percentage of the series examined per week varied from 0·1 to ·02 per cent. Another fallacy is that a weekly mean for one year has been taken. Now plague is most distinctly seasonal; one might as well attempt to express the snowfall of the British Isles in terms of departure from the weekly mean taken over every week of one year, including the summer months. The modified *M. rattus* theory, then, must go the way of the original one.

*Recent Conclusions of Plague Commission.*—Before passing on to Calcutta observations, I will say a few words as to the findings of the Commission in their last Report (*Journal of Hygiene*, May, 1908). Searchings would, perhaps, be a better word than findings. They have tried to establish that plague is associated with certain temperature conditions which are favourable to the retention of the plague bacillus in the stomach of the flea and favourable to the development of septicæmia in the rat, &c., and that these conditions are correlated to the mean temperature found to prevail in the seasonal period of plague activity. In the first place, they have failed completely to establish a mean temperature for plague epidemics. It is unnecessary to cite examples from their own records, as their failure can be best gauged from their own words: "*A plague epidemic, however, may come to an end when the temperature is most suitable*," p. 287. As regards their experiments on septicæmia and flea transmission at varying temperatures, it is difficult to make out which temperature they consider

the most suitable one, as in their final conclusion, p. 301, they find that a "suitable temperature is one somewhat below 85° F., and in general over 50° F." As a matter of fact, it does not matter which temperature they take as most suitable, as whatever they fix on within limits is upset by their own experiments. They obtained 100 per cent. of successes in flea transmission at 40°, 50°, 60°, and 70° F.; the last experiments were carried out in the non-plague months of July and August. At 90° F. 72 per cent. of twenty-seven rats developed septicæmia, and 33 per cent. showed abundant bacilli. These experiments and conclusions speak for themselves. The experiments are in absolute agreement with general clinical experience in India, and the conclusions are at variance with both experiments and experience.

*Calcutta Flea Conditions.*—My statements as to flea prevalence in Calcutta are based on the following observations:—

(1) Ten years' experience of the absence of fleas in connection with plague in Calcutta, corroborated by colleagues and subordinates. (2) A series of over 149 guinea-pig experiments extending over three epidemics, placed out as flea traps in presumably infected houses. In seventy-five of these houses the history was that of typical acute plague with buboes. (3) Flea counts on over 4,935 rats, extending over two epidemics. (4) Microscopic examination of over 1,338 fleas dehydrated and cleared. (5) Miscellaneous observations not recorded in statistics, including special enquiries at admission rooms of Calcutta hospitals. (6) Flea-biting experiments.

*Flea Prevalence in Calcutta.*—The rat-flea of Calcutta is as in Bombay, *Pulex cheopis*.[1] Other fleas are, practically, never found on the rat. *P. irritans* is not found at

[1] The latest name is *Læmopsylia cheopis*.

all. The counts, so far, show that the average per rat varies from about two in the later months of the year, the non-epidemic time, to about six in the epidemic time. Fleas on man are practically never found, an observation confirmed by Major Vaughan, I.M.S., and others in the admission wards of the hospitals. In fact, I have still to find my first *P. irritans* in Calcutta. This observation has been confirmed by Captain Gourlay, I.M.S., in Dacca. Except for a very few imported fleas (*P. irritans*) on the persons of hillmen, man is not a host for fleas in Dacca. *P. felis* or *canis*, the dog-flea, is occasionally found in an excessive prevalence, especially when dogs or cats have been removed. This, however, is mainly amongst Europeans and is in no way correlated to plague. There is no mistaking dog-flea prevalence, as I know from practical experience; the whole of the cane mat is alive with fleas, and the moment your naked foot touches the floor there is a black ring round your ankle. A lady confined to bed in the next room was very severely bitten, so that the discomfort and disfigurement were extreme. The importance of this observation you will gather later.

A large number of observations have been made as to the number of fleas found on wild rats, counts having been made on over 4,935 rats, some fully protected from the light, others not. It is unnecessary to give much detail as to these counts, as full particulars will be found in the tables at the end. They show that when precautions are taken to get a full count there is an increase in the numbers found in the epidemic season averaging about four to six as against about two in the off plague season. It is extremely difficult to come to any reliable generalization as to the average number on the various species or the margin of difference between the numbers

found on rats protected from the light and those which were not protected. For the thing that strikes one most is the great capriciousness of the variations found and the extreme contradictoriness of the results obtained. While fleas were increasing on a particular species in one ward, they might be decreasing in the next ward. The rats obtained from a particular source of supply might be almost flea-free for weeks and then suddenly for a few days fleas would be found in numbers, only to disappear again as rapidly as they had appeared. Another point on which there can be no doubt is that the tendency of the flea to leave the rat when it is exposed to light has been exaggerated. Many of our highest counts have been got on exposed rats, and Dr. Crake latterly gave up the precaution of bagging the rats at daybreak as the difference in the results was totally incommensurate with the enormous trouble involved. Probably the flea is very much less nocturnal than he is supposed to be; witness the innumerable biting experiments in tubes and otherwise that have been carried out during the day by all experimental observers. Man rests motionless at night, and therefore conditions are more favourable to his being attacked. In cases of acute flea infestation, as I know from the instance I have already quoted, and from other experiences, the daylight activity and feeding propensities of the flea may be quite marked. I have seen terrier pups at midday, in open sunlight, swarming with dog fleas.

So far, we have shown that flea conditions are quite different in Calcutta to those found in Bombay, that *Pulex irritans* is practically unknown, that *Ctenocephalus felis* is found only in connection with cats and dogs, and that though the rat-flea occurs on rats in numbers comparable to those found in Bombay, neither it nor any

other flea has been found in plague houses unless exceptionally and in insignificant numbers. This general observation has been confirmed by a series of definite negative observations carried out by means of guinea-pig flea-traps used with all the precautions recommended by Liston and the Commission. In all, 149 guinea-pigs have been put out overnight in presumably infected houses and in houses evacuated on account of the death of rats. The result has been totally different to that found in Bombay. There 275 guinea-pigs picked up 4,681 fleas, or an average of 17 each (*Journal of Hygiene*, vol. vii., pp. 436-445), whereas in Calcutta 149 guinea-pigs yielded 164 fleas, or an average of 1·08, though the counts included individual counts as high as 41, 18 and 15. One hundred and four of the experiments showed no fleas. Not a single guinea-pig contracted plague, and only 2 died, fifty-seven days after the experiment, with no signs of plague. It may be noted, on the authority of Mr. Haffkine, that these guinea-pigs are exactly the same as those used in Bombay, Calcutta being one of their sources of supply. In some of the 149 houses there was some doubt as to the diagnosis of plague, but in 75 there was none, a clear history of typical acute plague with bubo being obtained. The flea-count in those houses did not depart from the average. Of course, there was no delay in placing out the guinea-pigs, and in a few instances the animals were placed in the same room as the living patient. You may think that those results are so much at variance with the Bombay experiments that you have some difficulty in accepting them. In a recent paper I collected from the Commission's third report instances of fleas failing to infect guinea-pigs in nineteen instances where the fleas recovered totalled 520. The most marked instance was

in Parel, where 108 and 150 failed to convey plague to the first guinea-pigs, though when transferred they gave it to two other guinea-pigs. Again, in Dhand, out of twenty-four houses showing human plague only five proved infective to guinea-pigs, and none of those houses had furnished plague rats. Out of 32 guinea-pig experiments in the town 22 were negative, and 3 of the 10 positive were in one house. In Kasel only 3 guinea-pigs died out of 18, though the rat epidemic was marked—283 acute cases and 23 chronic. Thus it will be seen that the divergence between the Calcutta experiments and the later observations of the Commission is much less than might have been predicted from their early observations. Full details of our experiments will be found in the Calcutta Plague Report for 1908.

*Does the Rat-Flea bite Man?* (*P. cheopis*).—We now come to the crucial question, a question that answered in the negative destroys the fundamental principle of the rat-flea theory. The experiments that were carried out may seem to be rather bold in that fleas were taken direct from wild rats and put on men who were most of them not inoculated, but it must be remembered that in addition to a number of weighty theoretical considerations, which I have gone into very fully in the Calcutta Plague Report for 1907, there were very reassuring practical observations. Thus special enquiries at the end of the plague season showed that those engaged in regularly collecting rats showed no special tendency to contract plague, nor did the men engaged in handling the rats at the rat depots.

"*Flea-feeding Experiments.*—The fleas were obtained almost entirely from *Nesokia bengalensis* by the double chloroforming method which I have mentioned in my last paper on pulicides.[1] The species of the fleas

[1] Hossack, *Ind. Med. Gazette*, October, 1907, p. 366.

was not determined, but from the figures as to the preponderance of *P. cheopis* it is clear that they must all have been of this species with possibly one *P. felis*. The fleas were placed in enamelled mugs on the bottom of which was some damp sand, the top being covered with a plate of glass. In the pulicidal experiments it was noted that fleas kept in an empty test-tube or an empty mug were practically all dead in forty-eight hours, showing only four survivors out of sixty-eight in two instances, but it was found that the addition of damp sand made a great difference to the period of survival. It also seemed to make considerable difference to their power of jumping, for while no fleas managed to jump out of the empty mugs several were lost from the sanded mugs. At first, when the fleas had been reduced by death or loss to one or two, they were kept in a test-tube with damp sand as instanced by flea No. 10, which was in a test-tube from the third to the ninth day of starvation; in the later experiments they were put in tubes from the first day on which feeding was attempted. As to loss, in addition to that caused by jumping out some were lost by climbing up the damp sticky sides of the mug and several by slipping under an irregularity in the edge of the test-tube when they were being given an opportunity to bite; others were lost while separating the sand from the flea. These details are necessary as an explanation of the paucity of experiments in the later days of starvation. In order to eliminate the personal element as far as possible, for it is well known that some individuals are relatively exempt to attacks from fleas, eighteen individuals were used as subjects, and the same flea as far as possible was never put twice to the same person. On several occasions fleas having refused one person were at once put on another, but in only one case did this induce them to bite. The individuals used as subjects comprised, besides Mohommedans, the following castes of Hindus, Brahmin, Kyastha, Subernabanic, Dosad, Dome, Mehtar, and were drawn from the staff and the rat-catchers. I only used myself once or twice, as I am not very liable to attack from fleas. Some of the men had hairy arms, some had smooth.

"*Technique.*—The method adopted to isolate the flea in the tube was rather troublesome and tedious. The tube was placed on top of the flea as it sat on the sand; a bent spatula was then inserted in the sand under the tube, and thus the flea and a lump of sand were removed together. With some manipulation and the aid of a wire scoop the sand was then removed. The fleas were generally put to bite in pairs, and the site selected was the inner surface of the forearm where the skin is thin. At first the tube was left on for three to five minutes, but the period was latterly reduced, experience having shown that if a flea was going to bite it did so immediately it touched the skin. As a rule, the fleas

evinced a great desire to get off the skin and climbed up the tube and had to be repeatedly shaken down. It was sometimes observed that a flea would apparently settle down to bite as soon as he touched the skin, but would immediately change his mind either from distaste or inability to draw blood and would make no further attempt. When a flea did feed he bit as soon as he touched the skin, and remained sucking from two to three minutes, three and a half minutes being the maximum. Thrice a fresh site was selected after one and a half or two minutes and the feeding was renewed, but as a rule the feeding was uninterrupted. The total duration of the longest divided feed was five and a half minutes. Generally a very faint ecchymosis could be made out in light skins within five minutes of biting, sometimes so small that without the guide of the pressure mark of the test-tube it would be very difficult to find. On one occasion when the man had been bitten twice it was observed forty-eight hours after the bite that there were minute papules where he had been bitten. On one occasion the bite caused first a faint ecchymosis and within half an hour a white bleb $\frac{1}{8}$ inch by $\frac{1}{4}$ inch appeared. On this occasion the flea bit for only one and a half minutes, and no blood could be seen passing into it. It may be suggested that it was making violent and more or less unsuccessful efforts to suck, as the result of which there was an excessive salivary outflow. Blebs were only seen twice altogether.

"*Possible Criticism of Feeding Experiments.*—There are several points in these experiments in which it is desirable to meet objections which may reasonably be raised. First there is the objection that the conditions under which the experiments were conducted were unnatural and not conducive to biting. The answer to this is that the conditions are much less unnatural than those under which a number of successful biting experiments on rats and guinea-pigs were carried out by the Commission, for in these the fleas were not only applied in test-tubes, but had to bite through muslin. The fact that the experiments were made in the day and not at night is discounted in the same way by the successful biting experiments of the Commission carried out through the day, and also by the observations which I have already called attention to which tend to show that *P. cheopis* is not so purely nocturnal as it is supposed to be. The last factor that remains to be considered is the possible effect of the common custom in Bengal of applying to the body mustard or cocoanut oil. In the first place, oil was not applied by the Mehtars and low-class Mohommedans who were the subjects of the experiments. In the second place, even in those subjects who did apply oil there was no trace of it to be found either by touch or smell. The explanation of this is that only a very small quantity is applied; about a teaspoonful is put in the hand, the head is then well rubbed and only a scanty remainder is left for the trunk and limbs, and this is well rubbed in.

TABLE A.

TYPICAL EXAMPLES OF FLEA-BITING RECORDS. X = BITE, 0 = REFUSAL.

| Ser. No. | Date of capture | Date of first experiment | Days of starvation | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| 10 | 21/8/07 | 22/8/07 | 0 | 0 | 0 | – | 0 | 0 | 0 | 0 | dead |
| 19 | 22/8/07 | 23/8/07 | 0 | 0 | — | x | 0 | x | – | — | — |
| 21 | 26/8/07 | 27/8/07 | 0 | x | — | — | — | 0 | 0 | 0 | dying |
| 34 | 2/9/07 | 4/9/07 | — | 0 | 0 | 0 | 0 | — | 0 | 0 | dead |
| 51 | 4/9/07 | 6/9/07 | — | x | — | — | x | 0 | 0 | 0 | 0 |
| 52 | 4/9/07 | 6/9/07 | — | 0 | — | — | 0 | 0 | dying | — | — |
| 60 | 4/9/07 | 7/9/07 | — | — | 0 | — | 0 | 0 | lost | — | — |
| 63 | 4/9/07 | 7/9/07 | — | — | 0 | — | x | 0 | 0 | x | 0 feeble |
| 68 | 4/9/07 | 9/9/07 | — | — | — | — | x | 0 | x | 0 | 0 |
| 66 | 4/9/07 | 9/9/07 | — | — | — | — | 0 | 0 | 0 | 0 | 0 |

TABLE B

ANALYSIS OF RESULTS OF FLEA-FEEDING EXPERIMENTS.

| Day of starvation | Number of fleas per day of starvation | Number of bites | Ratio |
|---|---|---|---|
| 2nd | 41 | 1 | 1 in 41 |
| 3rd | 36 | 5 | 1 in 7 |
| 4th | 31 | 9 | 1 in 3½ |
| 5th | 28 | 5 | 1 in 5 |
| 6th | 21 | 6 | 1 in 3½ |
| 7th | 20 | 4 | 1 in 5 |
| 8th | 11 | 1 | 1 in 11 |
| 9th | 8 | 2 | 1 in 4 |
| 10th | 4 | 0 | 0 |
| Total | 200 | 33 | 1 in 6 |

For the 200 experiments 100 fleas were used; biting was confined to 26 out of the 100, and only 7 out of the 26 bit twice; 45 out of the 100 fleas had opportunities of biting twice. Of the 7 fleas that bit twice, all but one, No. 85, had refused more than once. Of the 26 fleas that bit all had refused once or oftener, except No. 85, and 5 fleas which had no chance of refusal after the first bite, Nos. 56, 71, 76, 87, 100.

One flea refused to bite at all in seven trials extending over nine days and seven different subjects. Another failed to bite after six trials on eight different subjects extending over nine days. The accidental losses were so numerous that I did not arrive at very definite conclusions as to

the duration of life in fleas that did bite. In the later days of the experiment I noted some as being very feeble in spite of having bitten once or twice. On the second day of starvation only one flea out of 41 would bite, but as starvation progressed bites became more frequent. Of opportunities to bite only 1 in 6 was taken, and of the fleas experimented with only 1 in 4 bit at all.[1]

*Acutely Infected Calcutta Houses.*—From the early experiments of the Commission I arrived at the conclusion that, where the flea counts were low, plague was either not transmitted at all or with difficulty. The Commission themselves have shown that one flea does not generally transmit plague (one success in sixty experiments) and admit that the rate of progress of an epizootic is conditioned by the number of fleas present. Verjbitski had noted in his own experiments what I have called attention to in the Commission's results and proceeded to define the limit. He found that even with very highly virulent plague "no infection is likely to take place when the number is less than five" (*Journal of Hygiene*, vol. viii., p. 185). Now the rat is more susceptible than man, and leaving out of account the enormous difference in bulk, man will probably require more bites, say ten. If my statement be accepted that of even starved rat fleas, only one in six will bite, then there must have been sixty starved fleas on man to produce the necessary ten bites in order to infect him. At the very least it is clear that there must be numerous fleas on him. Take now the cases of acutely infected houses in Calcutta, such houses as 19, Armenian Street, with twenty cases; 14, Roop Chand Roy Street, with twelve cases, and other houses with eight and ten cases all occurring within a few days. If these cases were due to flea-bites, then the houses should have been swarming

[1] Dr. Crake confirmed these experiments; only 8 bit out of 67 fleas starved up to twelve days.

with rat-fleas, something comparable to the dog-flea infestation I have already described, or the flea infestation repeatedly described in Bombay. No fleas were noticed in these houses, though the circumstances were peculiarly favourable to my detecting them had they been present. For I was at that time busily engaged in collecting clinical data, data I may observe which showed that in 30 per cent. of the cases, there was evidence of some involvement of the lung as is to be expected in a septicæmic disease, and in consequence I spent hours in these houses every day, often actually kneeling or reclining on the filthy platform beds on which the patients were lying. Yet I never saw a flea.

Conclusion.—*When these observations are taken in conjunction with our general experience in Calcutta and with the negative result of guinea-pig flea-traps, it is evident that rat-fleas can have played only an insignificant part in the acute plague which in certain wards of Calcutta attacked in a single epidemic* 1 *in* 40 *and* 1 *in* 30 *of the population.*

*Biting Experiments by the Commission.*—The Commission have carried out various experiments on the capacity of the flea to feed on man and bite him. It has already been pointed out that most of their early observations were carried out under artificial conditions or under exceptional circumstances such as those of 280, De Lisle Road, and even then referred not so much to fleas biting as to fleas being found on men. I also criticized the feeding experiments as being carried out in such a way as to make it impossible to get exact observations as to the number of bites. This criticism is to be found repeated by themselves in their last report. In experiment II., p. 251 (e) they admit it is a matter of considerable difficulty to know how many had fed. On

pp. 249-253, *Journal of Hygiene*, May, 1908, will be found a large series of experiments. In those very large numbers of fleas, which had been kept starved in boxes or go-downs, were given an opportunity of biting man. In some of the experiments, namely, II., III., II. (e) (1) and I., p. 252, it is possible to give definitely a minimum count. The fleas in these experiments exceeded 696 probably by a large margin, but only eleven bites were obtained, *i.e.*, only one in sixty-three bit and the fleas were starved. In the other experiments, starved fleas were present in very large numbers indeed—100 fleas per box is taken as a comparatively small count—but the proportion of bites was extremely small. From the figures given it cannot be stated accurately ; it varied from about 1 in 20 to 1 in 100. Is it unreasonable to maintain that these experiments absolutely support my contention that *Pulex cheopis* unstarved will hardly bite man at all and even starved only occasionally? If Verjbitski's conclusion, that even for the rat a minimum of five bites is necessary, be accepted, and in view of the Commission's results it must be, it follows, quite apart from plague in Calcutta, that the rat-flea can only play an occasional and unimportant part in human infection. It is a part comparable to the *rôle* of the human-flea and the cat-flea as established by Verjbitski, the *rôle* of flies, spiders and cockroaches as put forward by Hunter, and the *rôle* of the bed-bug as established by Verjbitski and quite recently by Jardanski and Kladnitsky. In fact, it seems to me that the whole position will have to be reconsidered.

This paper is already so lengthy that I cannot take up the various general arguments against the flea theory, nor the facts in favour of there being not one but many modes of plague infection. Moreover, I have already dealt with

them pretty fully in the Calcutta Plague Report of 1907. Amongst recent writers who are more or less in sympathy with my views may be mentioned Galli Valerio, Simpson, King, Klein, Baxter-Tyrie, and Hunter.

In conclusion, I should like to express my gratitude to the Commission, or rather to one of its leading members (Dr. Martin) for the courtesy and assistance he has so kindly shown me in arranging for me to carry on my work in this country in the plague laboratory in Elstree. He is only repeating what I have already experienced at the hands of Major Lamb in Kasauli. It is gratifying to find that acute controversial differences have only had the effect of establishing pleasant personal relations.

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# APPENDIX.

## TABLE I.

CRUDE FLEA COUNT, AUGUST-DECEMBER, 1906, BY DR. HOSSACK.

| | *M. decumanus* | | | *M. rattus* | | | *M. bengalensis* | | | *N. bandicota* | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Rats | Fleas | Max. | Rats | Fleas | Max. | Rats | Fleas | Max. | Rats | Fleas | Max. |
| August .. | 56 | 57 | 7 | 10 | 19 | 4 | 28 | 30 | 22 | 21 | 1 | — |
| September .. | 7 | 1 | — | 3 | — | — | 32 | 28 | 2 | 3 | — | — |
| October .. | 7 | 47 | 40 | 2 | 4 | — | 2 | 1 | — | — | — | — |
| November .. | 138 | 194 | — | — | — | — | 30 | 71 | — | — | — | — |
| December .. | — | — | — | — | — | — | 22 | 56 | — | — | — | — |
| | 208 | 299 | — | 15 | 23 | — | 114 | 186 | — | 24 | 1 | — |
| | *M. decumanus*, 1·42 fleas per rat. | | | *M. rattus*, 1·53 fleas per rat. | | | | | | | | |

Total rats all kinds, 337 = 508 fleas—1·50 fleas per rat.

CRUDE FLEA COUNTS, *N. bengalensis*.

| | | | Rats | | Fleas | | |
|---|---|---|---|---|---|---|---|
| Dr. Hossack. | June | .. | 21 | .. | 7 | .. | ·3 per rat |
| ,, | July | .. | 166 | .. | 392 | .. | 2·36 ,, |
| ,, | August | .. | 209 | .. | 284 | .. | 1·36 ,, |
| Dr. Crake. | August | .. | 216 | .. | 335 | .. | 1·55 ,, |
| | | | 612 | | 1018 | | 1·66 |

## TABLE II.

BAGGED NESOKIA FLEA COUNTS, 1907.

| | | | Rats | | Fleas | | Ratio |
|---|---|---|---|---|---|---|---|
| Dr. Hossack. | July | .. | 51 | .. | 107 | .. | 2·09 |
| ,, | August | .. | 108 | .. | 184 | .. | 1·70 |
| Dr. Crake. | August | .. | 216 | .. | 686 | .. | 3·18 |
| Special collection. | August | .. | 45 | .. | 139 | .. | 3·08 |
| | | | 420 | | 1116 | | 2·65 |

## TABLE III.

FLEA COUNTS BY DR. HOSSACK FOR 1908.

BAGGED COUNTS ON MIXED RATS, DISTRICT II.

| | | Rats | | Fleas | | Average |
|---|---|---|---|---|---|---|
| January | .. | 228 | .. | 908 | .. | 4·49 |
| February | .. | 168 | .. | 962 | .. | 6·7 |
| March | .. | 183 | .. | 1134 | .. | 6·5 |
| April | .. | 209 | .. | 1280 | .. | 6·8 |

COUNTS ON UNBAGGED MIXED RATS, DISTRICT II.

| | | Rats | | Fleas | | Average |
|---|---|---|---|---|---|---|
| January | .. | 103 | .. | 255 | .. | 2·4 |
| February | .. | 126 | .. | 385 | .. | 3·0 |
| March | .. | 64 | .. | 173 | .. | 2·1 |
| April | .. | 12 | .. | 43 | .. | 3·7 |

FLEA COUNTS ON UNBAGGED RATS, DISTRICT III., BY SPECIES.

| | *M. decumanus* | | | *M. rattus* | | | *N. bengalensis* | | | *Mixed rats* | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Rats | Fleas | Av. | Rats | Fleas | Av. | Rats | Fleas | Av. | Rats | Fleas | Av. |
| January .. | 18 | 26 | 1·5 | — | — | — | 35 | 65 | 1·8 | 5 | 12 | 2·4 |
| February .. | 50 | 276 | 5·5 | 2 | — | 1·5 | 152 | 415 | 2·7 | 62 | 181 | 2·9 |
| March .. | 9 | 62 | 6·7 | — | — | — | 145 | 706 | 4·8 | 29 | 121 | 4·1 |
| April .. | 6 | 95 | 15·8 | — | — | — | 89 | 376 | 4·2 | 18 | 91 | 5·0 |

## FLEA COUNTS IN DISTRICT I. BY DR. CRAKE.

### FIRST SERIES, 1906-07.

Average number of fleas per rat, all types, all seasons of year, 1·19 (219 fleas on 183 rats).

Average number of fleas per rat, all types: August to December, 1·3; January to March, 1·5.

*Mus rattus.*—Average, August to December, 1·7; January to March, 3·0 (numbers small, not recorded till confirmed by later observations).

### SECOND SERIES, 1908, QUIESCENT PERIOD.

(*a*) Bagged *N. bengalensis*, 271 rats gave 803 fleas, average 2·9 per rat (between July 16, 1907, and September 13, 1907).

(*b*) Unbagged 214 *N. bengalensis* gave 335, average 1·5 per rat.

Up to December no increase at north of town.

Certain rats from Ward V yielded large numbers: 92 *N. bengalensis* giving 335 fleas, average 4·3 per rat.

In District I., December, 151 *Nesokia* gave 174 fleas, average 1·1 per rat. January, 137 *Nesokia* gave 391 fleas, average 2·9 per rat. (Ten from Ward V gave 54 fleas).

In February separate counts for each species made. Following table is for February to June, 1908:—

## TABLE IV.

FLEA PREVALENCE. DR. CRAKE, 1908.

*Unbagged Counts.*

| Month | Species of rats | Rats | Fleas | Average |
|---|---|---|---|---|
| February .. | *M. rattus* .. .. | 81 .. | 596 .. | 7·2 |
| ,, .. | *M. decumanus* .. | 37 .. | 302 .. | 8·1 |
| ,, .. | *M. bengalensis* .. | 353 .. | 1,011 .. | 2·8 |
| ,, .. | All types .. .. | 471 .. | 1,909 .. | 4·05 |
| March .. | *M. rattus* .. .. | 8 .. | 31 .. | 3·8 |
| ,, .. | *M. decumanus* .. | 33 .. | 365 .. | 11·06 |
| ,, .. | *M. bengalensis* .. | 326 .. | 767 .. | 2·3 |
| ,, .. | All types .. .. | 367 .. | 1,163 .. | 3·1 |
| April .. | *M. rattus* .. .. | *Nil* .. | *Nil* .. | *Nil* |
| ,, .. | *M. decumanus* .. | 31 .. | 383 .. | 12·3 |
| ,, .. | *M. bengalensis* .. | 305 .. | 610 .. | 2·0 |
| ,, .. | All types .. .. | 336 .. | 993 .. | 2·9 |
| May .. | *M. rattus* .. .. | 4 .. | 11 .. | 2·7 |
| ,, .. | *M. decumanus* .. | 11 .. | 75 .. | 6·8 |
| ,, .. | *M. bengalensis* .. | 475 .. | 337 .. | 0·7 |
| ,, .. | All types .. .. | 490 .. | 423 .. | 0·86 |
| June .. | *M. rattus* .. .. | 14 .. | 12 .. | 0·8 |
| ,, .. | *M. decumanus* .. | 5 .. | 4 .. | 0·8 |
| ,, .. | *M. bengalensis* .. | 270 .. | 111 .. | 0·41 |
| ,, .. | All types .. .. | 289 .. | 127 .. | 0·43 |

# A SHORT ABSTRACT OF THE PLAGUE COMMISSION'S WORK IN BOMBAY WITH REGARD TO THE RAT-FLEA THEORY.

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I wish in the first place to thank the Council of this Society for their courtesy in inviting me to take part in the discussion on Dr. Hossack's paper. Dr. Hossack has brought forward to-night some criticisms against the rat-flea theory of plague. Since his criticisms against this theory have derived their inspiration in no small measure from the published reports of the Plague Research Commission it may be of interest if I, as one who worked with the Commission in Bombay, attempt to summarize briefly the evidence on which the members of the Commission base their conclusions.

At the present time most plague workers accept the view that the rat epizootic has a very important association with the human epidemic. Now this takes us a long step forward towards a general agreement. Difficulties, however, arise when we begin to discuss the exact mode of infection of man by plague-rats, and the possibility of modes of infection of man other than by rats. In order to make matters quite clear at the outset, let me define the position of the Commission on this point. As I understand it, it is this :[1] They maintain that the essential agent in the transmission of plague to man is the rat-flea. They do not deny the possibility of other

[1] Note.—The final conclusions of the Commission have not yet been officially published.

methods of transmission of infection being effective, but they maintain that these methods are so rarely effective as to be quantitatively negligible from an epidemiological standpoint. They hold, in short, that the essential fact in the epidemiology of plague is that the infection of man is brought about in the overwhelming majority of instances by the agency of the rat-flea. It rests, of course, with the Commission to give good grounds for this conclusion, and I want to say also that it rests with their critics to produce equally good or better evidence of an opposite character derived from their own observations. In this connection, may I point out that there are three methods of criticism, two of which have been somewhat liberally employed by the opponents of the views of the Commission. Stated inversely in their order of merit these are: First, the method of purely academic criticism unsupported by personal observation, which consists in the discovery of seeming inconsistencies in an opponent's case. Much of Dr. Hossack's criticism of the Commission's work is undoubtedly of this character, and is not, therefore, strictly speaking, valid. There is, again, criticism not based on personal observation but which consists in invoking the aid of other workers who have arrived at conclusions which do not accord with those of one's opponent. This, for obvious reasons, is a form of criticism which ought to be used with discretion. There is, lastly, the true critical method—the use of observational and experimental methods, and the comparison of results obtained by these methods with those obtained by other workers.

The two principal problems in the epidemiology of plague are: (1) The mode or modes of infection of rats in an epizootic of plague, and (2) the mode or modes of plague-infection in man. I shall state under each of these

headings the conceivable modes of infection, and shall briefly adduce the Commission's evidence for or against each possible method of transmission.

## Modes of Infection in Rats.

There are three possible modes of infection in rats, namely, through the skin, the lungs, and the alimentary tract.

(1) *Intestinal Infection in Rats.*—It is conceivable that rats in nature are infected by feeding on the carcasses of plague-infected rats, or on food contaminated by the excreta of plague-rats. What is the Commission's evidence for or against this method of transmission? A large number of Bombay rats were fed with plague-infected material. As a result a number of them died of plague, and in these mesenteric and neck buboes were commonly found. The proportion of mesenteric to neck buboes was two to one. Taken by themselves these results have little or no significance. But they were supplemented by the examination of a very large number of rats found dead of plague in the streets and gullies of Bombay. Out of 5,188 plague-rats 72 per cent. had buboes in the neck, but in no single instance was a mesenteric bubo found. The Commission examined nearly 18,000 plague-rats in Bombay throughout a year's investigation, and never once found a rat with a primary mesenteric bubo. The Commission, I think, are justified in concluding from these facts that the naturally infected rats are not infected by feeding. Now certain writers, including Professor Simpson and Dr. Hossack, have pointed out as an argument against this conclusion, that a number of other observers, *e.g.*, the Russian, Austrian, and German Commissions, Kolle, Klein, Kister and

Schumacher, have succeeded in infecting rats in the laboratory by feeding them on plague material, and have suggested accordingly that rats in nature are infected in this way. This, surely, is a fallacious method of reasoning, simply because these authors have failed to control their experimental results by observations on naturally infected rats. In the whole literature of plague I can find only two adequate descriptions of the *post-mortem* appearances of naturally infected rats. The Austrian Commission describes the lesions in eight plague-rats, and Skschivan, in Odessa, those of thirty-two plague-rats. Let me state a parallel case to the one we are considering. It is possible to infect guinea-pigs in the laboratory by feeding them with tuberculous material. Would it be justifiable from this result alone to conclude that guinea-pigs spontaneously infected with tubercle are infected by way of the intestine?

(2) *Infection through the Lungs.*—The evidence regarding this mode of infection is conclusive, but has no special significance in relation to the epizootic. 0·6 per cent. of naturally infected Bombay rats show a definite lobar pneumonia.

(3) *Skin Infection in Rats.*—It is conceivable that rats in nature may be infected by the accidental inoculation of infected rat excreta into abrasions. The Commission inoculated the fæces of plague rats cutaneously into fresh rats; only about 3 per cent. of the latter died of plague. The urine of plague rats was found to contain much fewer plague bacilli than the blood; thus while the blood of a rat may have as many as 100,000,000 organisms in a cc. the urine may have none at all, or at least less than 10 per cc. This evidence does not suggest that the excreta of plague-rats plays any important part in the spread of the epizootic.

It is possible that rats in nature may become infected by rat-fleas. The Commission's evidence on this point is presented summarily in the accompanying table, which shows that the regional distribution of buboes in naturally infected rats and in rats experimentally infected by fleas, is practically the same.

SHOWING THE PERCENTAGE FREQUENCY OF BUBOES IN DIFFERENT PARTS.

| | Naturally infected | Infected by fleas | Infected by feeding |
|---|---|---|---|
| Rats examined .. .. | 5,188 | 117 | 119 |
| Rats without buboes .. | 16 per cent. | 18 per cent. | 8 per cent. |
| Cervical bubo .. .. | 72 | 61 | 36 |
| Axillary ,, .. .. | 23 | 22 | 1 |
| Inguinal ,, .. .. | 18 | 35 | 1 |
| Mesenteric bubo.. .. | 0 | 0 | 72·5 |

Summing up the whole of the evidence on the question of rat infection, we may conclude that the rat-flea is the essential agent of transmission of plague from rat to rat in an epizootic.

## MODES OF INFECTION IN MAN.

Just as in the case of the rat, the conceivable paths of infection in man are the skin, the lungs and the intestines.

(E) *Infection through the Skin.* — (*a*) *Inoculation of Infected Human Excreta into Abrasions.*—It is possible that the attendants of a plague patient may contract the infection by the accidental inoculation of his infected excreta or secretions, pus, mucus, &c. The Commission tested the infectivity of the urine and fæces of a number of plague patients, acutely ill of the disease, by the method of cutaneous inoculation into guinea-pigs. The Bombay guinea-pig, it may be remarked, is an extremely sensitive indicator of plague infectivity. The fæces were found to be rarely infective. Fifty-seven samples of urine from thirty-seven cases, eleven of which contained plague

bacilli (19·3 per cent.), were tested in this way, but failed to communicate the disease. Again, guinea-pigs were exposed for two or three weeks to intimate contact with bedding recently contaminated with the dejecta of acute cases, but all remained healthy. The general experience with regard to plague hospitals that the attendants on the inmates are singularly free from danger of infection, was fully verified by the Commission in Bombay. Moreover, a number of "imported" plague cases, *i.e.*, cases brought into a plague-free district from an infected district, were investigated by the Commission. In no instance did the infection appear to spread from the plague patient to other occupants of the house. Lastly, the figures relating to the occurrence of single and multiple cases in houses in Bombay were analyzed for several epidemics. This analysis conclusively showed that the occurrence of single cases in houses is the rule.

(*b*) *The Inoculation of Infected Rat Excreta into Abrasions.*—If human excreta are not effective in spreading the disease it is difficult to conceive how rat excreta can be effective in this regard.

(*c*) *The Transference of Infection from a Human Case by means of the Human-flea, Mosquitoes, Bugs, or other Insects.*—The relatively slight septicæmia in human plague compared with rat plague would diminish the chances of these insects becoming infected by imbibing human blood. Moreover, the evidence relating to the spread of infection by direct contact already adduced, bears against the view that these insects play any important part in the spread of the epidemic.

(*d*) *The Rat-flea as an Agent of Transmission of Infection from Rat to Man.*—The Commission have shown by numerous experiments that infected rat-fleas may occasion severe epidemics amongst susceptible animals, viz.,

guinea-pigs—indeed they were unable to produce experimental epidemics by any other means—and that when guinea-pigs are placed in plague-houses the method of infection is undoubtedly the rat-flea. They are led to the latter conclusion by a comparison of the regional distribution of buboes in guinea-pigs experimentally infected by fleas and in guinea-pigs which have contracted their infection in plague-houses. The percentage figures are shown in the following table.

SHOWING THE PERCENTAGE FREQUENCY OF BUBOES IN DIFFERENT PARTS.

| | Naturally infected | Infected in go-downs | Infected by fleas |
|---|---|---|---|
| Guinea-pigs examined .. | 87 | 253 | 108 |
| Without buboes .. .. | 1 per cent. | 1 per cent. | 1 per cent. |
| Cervical bubo .. .. | 92 | 92 | 91 |
| Axillary ,, .. .. | 1 | 3 | 6 |
| Inguinal ,, .. .. | 14 | 13 | 22 |
| Mesenteric bubo .. .. | 0 | 0 | 0 |

These results may with reason be utilized to explain the spread of the human epidemic, provided it be shown that the chances are considerable of man being bitten by rat-fleas during the epidemic period, and that the chances of man becoming infected as the result of their bites are considerable. In this connection it may be noted that the Commission have shown (1) that the rat-flea, especially if starved, will come on to and bite man; (2) that even in the absence of plague-infection rat-fleas are more numerous in the native houses in Bombay during the epidemic season than during the non-epidemic season; (3) that in the epidemic season houses which were definitely proved to be plague-infected contained on an average nearly three times the number of rat-fleas contained in houses which were only presumably plague-infected, and twelve times the number contained in houses which were free from suspicion; (4) that the percentage of fleas that have been taken from septicæmic

plague-rats found with abundant plague bacilli in the stomach contents varies with the season of the year, being six times greater in the epidemic season than in the non-epidemic season; (5) that 32 per cent. of rat-fleas (41 out of 130 dissected) caught in plague-houses in Bombay were plague-infected; (6) that the fæces of infected fleas frequently contain plague bacilli in enormous numbers—probably amounting to many millions.

If, then, an infected rat-flea bites man, owing to the habit of the flea of ejecting several droplets from the rectum during the act of imbibing blood, many millions of plague bacilli will be deposited on the skin at a distance from the puncture wound roughly corresponding to the length of the flea's body. It is not surely difficult to imagine that many bacilli may be rubbed into the wound by the scratching occasioned by the irritation of the bite. The Commission have, in fact, demonstrated that the puncture wounds made by rat-fleas permit the entrance of plague bacilli in sufficient numbers to cause a fatal infection in susceptible animals, and that the fæces of fleas taken from plague rats are infective to susceptible animals both by cutaneous and by subcutaneous inoculation.

(II.) *Infection through the Lungs.*—The percentage of pneumonic cases appears to vary in different outbreaks. Choksy estimates the percentage in Bombay at 1 per cent.

(III.) *Intestinal Infection.* (*a*) *From Food contaminated by Infected Human or Rat Excreta.*—In the first place, the plague bacillus is a delicate organism and does not thrive well as a saprophyte. The infectivity of rat excreta, as already noted, appears to be slight. The Commission's contact experiments with guinea-pigs in flea-free go-downs show that susceptible animals do not contract the disease

even when they eat food contaminated with the excreta of plague-infected companions.

The members of the Austrian Plague Commission in their classical memoir on the pathology of human plague make this definite statement: "Undoubtedly in the overwhelming majority of instances the infection occurs through the skin. In no single instance did anything point to an intestinal infection."

Again, Choksy, of Bombay, who has had exceptional opportunities for observing plague in its clinical aspects, referring to the views of Drs. Hunter and Hossack on this matter, remarks in a recent report: "So far as our clinical and *post-mortem* observations go on this side of India, I am fully convinced that infection does not take place in the way suggested. In not a single instance among over one hundred autopsies at the Arthur Road Hospital have we come across evidence of a primary abdominal infection; the pathological changes noted in the abdominal cavity have always been secondary."

(*b*) *Plague in Domestic Animals as a Source of Alimentary Infection to Man.*—The Commission failed to find any evidence whatever to support the view that man may become infected by this means.

Summing up the Commission's evidence on the question of the infection of man, it is, in my view, legitimate to conclude (1) that leaving out of consideration the rat-flea all the methods of infection indicated above fail to explain the epidemic spread of the disease; (2) that viewing as a whole the mass of the Commission's evidence relating to the infecting power of the rat-flea and the occurrence of infected rat-fleas in plague-houses during the epidemic seasons the infection of man by the rat-flea must be regarded not only as a conceivable, but as a credible and adequate, explanation of the epidemic spread of the disease.

There is only one other point to which I wish to refer, and that is the question of the seasonal prevalence of plague. If the Commission's conclusions are correct, we must expect that this phenomenon is explicable on the view that the rat-flea is the essential agent of infection in the epizootic and epidemic. As a matter of fact, there is a marked prevalence of fleas in Bombay, and the season of flea prevalence is exactly that of the rat and human plague prevalence. It is a remarkable circumstance that in two places not far distant from each other the seasonal prevalence may be quite dissimilar. Thus, in Bombay plague begins to be epidemic in December and January, in Poona 80 miles distant and in Belgaum 250 miles distant from Bombay, plague begins in July and August. An investigation is at present being conducted in Poona and Belgaum with the object of determining whether there is a definite season of flea prevalence and whether if so this season corresponds with that of the rat and human plague prevalence. I am indebted to Dr. Martin, of the Lister Institute, for the information, that although the investigation is not yet complete, the results so far strongly indicate that a season of flea prevalence does exist in these places and that it coincides with the plague season.

Dr. Hossack's experiments with rat-fleas in Calcutta appear to have been on the whole negative. This is perhaps unfortunate from the point of view of the rat-flea theory so far as Calcutta is concerned. I wish to point out, however, that Dr. Hossack has omitted unaccountably to make some observations of crucial importance- -at least he has given us no record of them. If the rat-flea theory does not hold good in Calcutta, then rats become infected there by some other means, possibly by feeding. If this is so, it is equally unlikely that the human inhabitants of

Calcutta are infected by the rat-flea. It seems to me, therefore, that an investigation of the *post-mortem* appearances of at least 1,000 plague-infected Calcutta rats is urgently required; special attention being paid to the situation of the peripheral buboes, and the presence or absence of mesenteric buboes. In the absence of accurate statistics obtained in this way, opinions as to the likely modes of infection either in rats or in men in Calcutta, are worthless. If it is found that in a series of 1,000 plague-rats examined during the epidemic period, 70 per cent. showed mesenteric buboes, then I should frankly admit that the rat-flea theory, so far as Calcutta is concerned, is in Huxley's phrase, a beautiful theory killed by an ugly little fact. I await the evidence on this crucial point with some confidence as to the results. Until this evidence is forthcoming I am not in the least disposed to regard seriously Dr. Hossack's sceptical attitude towards the rat-flea theory of plague.

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## Discussion.

Sir Patrick Manson, commenting on Dr. Hossack's paper, said it was very gratifying to find that there were some members of the profession who had the courage to differ from accepted theories. He thought they were very useful members of the community and ought to be congratulated. What they had heard from Dr. Hossack to-night should lead to an animated discussion, and might possibly emphasize the value of the position that he had taken up. He confessed that after reading the reports of the Commission he had been convinced of the truth of the rat-flea theory of plague, but after hearing Dr. Hossack he began to waver in his belief. He did not suppose the rivalry which was known to exist between

both sides of the Indian Peninsula would influence the scientific opinion of a member of the profession of the qualifications and standing of Dr. Hossack, and he presumed that they must make up their minds to hesitate before accepting the opinions held in Bombay.

Sir HAVELOCK CHARLES said he had listened with very great pleasure to the lecturer and also to Dr. Petrie. He had previously read with much interest Dr. Hossack's paper on recent developments in the theory of plague infection. To-night the subject had been dealt with largely from the point of view of statistics, and he had heard statistics on both sides. He did not like them, and felt inclined to say, "a plague on both their houses"; he liked things put more plainly. Dr. Hossack's criticisms were highly trenchant, and they almost made him form the conclusion that, if Dr. Hossack be right, then the members who formed the Plague Commission (whose names were very distinguished) were either incompetent to carry out experiments, or incompetent to draw conclusions therefrom. Dr. Hossack deserved every credit for having backbone enough to oppose conclusions that he did not approve of, but it seemed to him that in India he was more or less like "a voice crying in the wilderness." One thing that struck him in Dr. Hossack's written paper was his drawing so much attention to No. 3 of the general conclusions of the pamphlet on the etiology and epidemiology of plague written by Major Lamb, I.M.S. Dr. Hossack charges the Advisory Committee with responsibility for the statement that plague is solely spread by the flea. This is absolutely contrary to fact, for the Advisory Committee have no responsibility for the pamphlet in question, or the statements therein. The only official *dicta* of the Advisory Committee are to be found in the Plague numbers of the *Journal of Hygiene*,

and nowhere else. In the last paragraph of the preface to the pamphlet it was distinctly laid down that the summary had been compiled by Major Lamb, who was responsible for the form in which the observations were set forth and the way in which they might be interpreted.

His (Sir Havelock Charles's) experience in plague dated from 1898. He happened to be the one who first diagnosed plague in Calcutta. He took Professor Simpson to one of the houses in which plague was, and in that house Professor Simpson was met by a sick rat. That rat was examined and found to be infected. These were days when it was not wise to say there was plague in Calcutta. The commercial bias against a diagnosis of plague was very strong, but in spite of that bias plague was declared and has remained there. From that date he had always looked for the rat, and he undoubtedly thought the association of rats with plague was practically almost invariable. If there were an exception there was generally something to explain that exception. He would suggest that in Calcutta plague had never made great headway, nor in Madras. The flea was not common there, but the bug was. In Mysore and in the Punjab, on the contrary, fleas were plentiful. In an empty house once, in the Punjab, he was overrun with fleas and could pick them up in handfuls. He merely suggested that possibly the reason why Calcutta had had a certain amount of immunity was that fleas are less common there than in certain other places. Dr. Hossack stated that the rat-flea did not bite. Here was a point in which he was a direct opponent of investigators in Bombay.

Sir Havelock Charles cited an instance from ancient history which occurred 3,000 years ago, in the neighbourhood of Egypt, where plague was always common. In

Ashdod there was a plague which was attributed to the presence of the Ark of the Covenant. The Ark was removed to Gath, and the plague followed it. From Gath it was taken away to Ekron; the plague followed it there. His contention was that the cover of the Ark, which was of badgers' skins, carried fleas, and that the fleas were the cause of infection.

With regard to the objections to the rat-flea theory, he thought that they came best from those who performed experiments direct, and although one could only criticize in a very feeble way these variable points, it was exceedingly interesting to do so. He would like to have Dr. Hossack's opinion upon that historical incident he had mentioned.

Dr. G. C. Low wished to ask one or two questions. First he enquired of Dr. Petrie why he laid so much stress on the presence of the cervical buboes in naturally infected rats. Why should 72 per cent., of the dead rats, picked up in plague-houses, have buboes in the neck? Secondly, why should you always expect mesenteric buboes in feeding experiments? Was it not possible for the mucous membrane of the neck and throat to be infected? Why should there be such a difference in the site of the buboes in feeding experiments? He thought some German investigators in their experiments showed conclusively that buboes in the neck were found. Thirdly, in the experiments referred to by Dr. Hossack (where he put the guinea-pigs in the houses), how did he afterwards catch the fleas? Manifestly the flea is such a small and agile thing that it was difficult to observe whether they were all caught. He was unable to see how quite accurate counts were made.

Mr. J. Cantlie thought that the question that should

be put down for solution was the primary evidence of the disease. We were blaming the rat whereas perhaps the rat would blame man. He did not think such a thing as bubonic plague had been found on the Gold Coast, Glasgow, and some other places; it was mostly pneumonic in type. Pneumonic plague was so easily conveyed from man to man. One could easily imagine infection of the lung developing itself now and again into septicæmic plague, or developing itself into the peculiar form called pneumonic plague. From what he had heard from Dr. Hossack's paper it seemed that we were neglecting the theory of transmission from man to man in plague.

Mr. H. G. Waters confirmed Mr. Cantlie's opinion about pneumonic plague being often passed on from man to man. In several large towns in India there was hardly a case of bubonic plague, the type was chiefly pneumonic.

Dr. Hossack in reply, said he thought Dr. Petrie and himself must agree to differ. Dr. Petrie had been dealing with modes of infection in rats. He, Dr. Hossack, had been concerned with the mode of infection in man. He was in agreement with most of what Dr. Petrie said as to conclusions being based on personal experiments. In Calcutta they had no plague laboratory, and he had had to work without a laboratory. This would explain why he had brought forward no positive theories—he was not yet thoroughly convinced in his own mind. One point to which he wished to refer was that Dr. Petrie gave the percentage of the cases of pneumonic plague in Bombay as 1 per cent.; Dr. Choksy read a paper some nine months ago and said that the proportion of pneumonic plague had been under-estimated. In reply

to Sir Havelock Charles, he was afraid he could hardly meet him on equal terms. He wondered on what grounds he had put forward his theory of the historical incident mentioned. He (Dr. Hossack) had had no personal experience with the Ark. A question was put by Dr. Low as to the method adopted in the counting of the rat-fleas on guinea-pigs. The procedure was very simple. The guinea-pigs were chloroformed lightly, and the fleas, being slightly affected too, were then easily combed out. A consecutive series of 760 rats were chloroformed by him deeply so that the rats died. The whole cage was then shaken over a sheet of white paper and the fleas collected and counted. Each individual dead rat was then treated similarly, and it was found that the difference of count was not more than 5 per cent.

Dr. PETRIE, in answer to Dr. Low's question as to why the proportion of mesenteric buboes was greater than cervical, said cervical buboes in feeding rats were due to injuries in the mouth and throat, and in the other case it simply meant that the infection got down the throat into the intestines, and that was why there should be so many mesenteric buboes in feeding experiments. As to why there should be so many cervical buboes in naturally infected rats, he would state that in the guinea-pigs which were infected with fleas, the fleas were counted in different parts of the guinea-pig and it was found that a larger number of fleas could be picked off from the neck than elsewhere. Perhaps the skin may be thinner about the neck, and this may account for it, but there was no definite evidence why cervical buboes should be so common in naturally infected rats.